Turner, S.
09/867847

FILE 'REGISTRY' ENTERED AT 12:14:51 ON 06 JAN 2003
L1 4616 S KLVF/SQSP

FILE 'HCAPLUS' ENTERED AT 12:15:16 ON 06 JAN 2003
L1 4616 SEA FILE=REGISTRY ABB=ON PLU=ON KLVF/SQSP
L2 3135 SEA FILE=HCAPLUS ABB=ON PLU=ON L1
L3 1359 SEA FILE=HCAPLUS ABB=ON PLU=ON L2 AND (AD(S)ALZHEIMER? OR ALZHEIMER? OR AMYLOID(S)(DISEAS? OR DISORDER))
L4 622 SEA FILE=HCAPLUS ABB=ON PLU=ON L3 AND (TREAT? OR THERAP? OR PREVENT? OR CONTROL?)
L5 29 SEA FILE=HCAPLUS ABB=ON PLU=ON L4 AND (VACCIN? OR IMMUNIS? OR IMMUNIZ?)

L1 4616 SEA FILE=REGISTRY ABB=ON PLU=ON KLVF/SQSP
L2 3135 SEA FILE=HCAPLUS ABB=ON PLU=ON L1
L6 164 SEA FILE=HCAPLUS ABB=ON PLU=ON L2 AND ((AD(10A)ALZHEIMER? OR ALZHEIMER? OR AMYLOID(3A)(DISEAS? OR DISORDER))(5A)(TREAT? OR THERAP? OR PREVENT? OR CONTROL?))
L7 17 SEA FILE=HCAPLUS ABB=ON PLU=ON L6 AND (VACCIN? OR IMMUNIS? OR IMMUNIZ?)

L8 29 L5 OR L7

L8 ANSWER 1 OF 29 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:927177 HCAPLUS
TITLE: **Amyloid** .beta. peptide fragment linked to helper T cell epitope for **prevention** and **treatment** of **Alzheimer**'s **disease**
INVENTOR(S): Wang, Chang Yi
PATENT ASSIGNEE(S): United Biomedical, Inc., USA
SOURCE: PCT Int. Appl., 77 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002096350	A2	20021205	WO 2002-US10293	20020402

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2001-865294 A 20010525
AB The present invention relates to a compn. comprising a peptide

immunogen useful for the **prevention** and **treatment** of **Alzheimer**'s Disease. More particularly, the peptide immunogen comprises a main functional/regulatory site, an N-terminal fragment of Amyloid .beta. (A.beta.) peptide linked to a helper T cell epitope (Th) having multiple class II MHC binding motifs. The peptide immunogen elicit a site-directed immune response against the main functional/regulatory site of the A.beta. peptide and generate antibodies, which are highly cross-reactive to the sol. A.beta.1-42 peptide and the **amyloid** plaques formed in the brain of **Alzheimer**'s **Disease** patients. The antibodies elicited being cross reactive to the sol. A.beta.1-42 peptide, promote fibril disaggregation and inhibit fibrillar aggregation leading to immunoneutralization of the "sol. Asz-derived toxins"; and being cross-reactive to the amyloid plaques, accelerate the clearance of these plaques from the brain. Thus, the compn. of the invention comprising the peptide immunogen is useful for the **prevention** and **treatment** of **Alzheimer**'s Disease.

IT **109770-29-8P 477826-92-9P 477826-93-0P 477826-96-3P**
RL: BSU (Biological study, unclassified); PRP (Properties); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(**amyloid** .beta. peptide fragment linked to helper T cell epitope for **prevention** and **treatment** of **Alzheimer**'s **disease**)

L8 ANSWER 2 OF 29 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:864789 HCAPLUS
DOCUMENT NUMBER: 137:324199
TITLE: Modeling **Alzheimer**'s **disease** immune **therapy** mechanisms: interactions of human postmortem microglia with antibody-opsonized **amyloid** beta peptide
AUTHOR(S): Lue, Lih-Fen; Walker, Douglas G.
CORPORATE SOURCE: Sun Health Research Institute, Sun City, AZ, 85351, USA
SOURCE: Journal of Neuroscience Research (2002), 70(4), 599-610
CODEN: JNREDK; ISSN: 0360-4012
PUBLISHER: Wiley-Liss, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The induction of an antibody response to **amyloid** .beta. (A.beta.) peptide has become a strategy for the **treatment** of **Alzheimer**'s **disease** (**AD**). This has proven effective in reducing the plaque burden in transgenic mice that develop A.beta. plaques similar to human AD patients. The mechanism for enhanced clearance of A.beta. is partly due to the interaction of Ig Fc.gamma. receptor-expressing microglia and specific antibody-opsonized A.beta. deposits. This interaction can stimulate Fc.gamma. receptor-mediated phagocytosis, but also results in inflammatory activation of these cells. Consequently, interaction of microglia with antibody-antigen complexes could exacerbate the existing inflammation in the brains of AD patients. Here, the authors used substrate-bound A.beta. and cultured human microglia from AD and non-demented cases to model interaction of

microglia and antibody-opsonized plaques in AD brains. Enhanced prodn. of tumor necrosis factor-.alpha., macrophage colony stimulating factor, interleukin-10, and superoxide ions was detected. The authors also demonstrated enhanced uptake of opsonized A.beta. by microglia, which was reduced in the presence of excess IgG, indicative of the involvement of Fc.gamma. receptor-mediated mechanisms. Human microglia were shown here to express mRNA for Fc.gamma. receptors I, IIa, IIb, and III. The expression of Fc.gamma. receptor II was augmented by proinflammatory stimulation. Thus, initial interactions of human microglia with antibody-opsonized amyloid could result in increased inflammation. The consequence of this on inflammatory pathol. in AD brains needs to be considered before **immunization** is used as a strategy for **treating** AD.

IT **107761-42-2, Amyloid** .beta.(1-42)
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(antibody-opsonized; Fc.gamma. receptor-mediated human postmortem microglia interaction with antibody-opsonized **amyloid** .beta. peptide in **Alzheimer**'s **disease** model in relation to inflammation induction and immunotherapy)

REFERENCE COUNT: 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 3 OF 29 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:831601 HCAPLUS
DOCUMENT NUMBER: 138:3442
TITLE: Generation of antibodies specific for .beta.-**amyloid** by **vaccination** of patients with **Alzheimer disease**
AUTHOR(S): Hock, Christoph; Konietzko, Uwe; Papassotiropoulos, Andreas; Wollmer, Axel; Streffer, Johannes; von Rotz, Ruth C.; Davey, Gabriela; Moritz, Eva; Nitsch, Roger M.
CORPORATE SOURCE: Division of Psychiatry Research, University of Zurich, Zurich, Switz.
SOURCE: Nature Medicine (New York, NY, United States) (2002), 8(11), 1270-1275
CODEN: NAMEFI; ISSN: 1078-8956
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English

AB To characterize antibodies produced in humans in response to A.beta.42 **vaccination**, the authors carried out immunohistochem. examns. of the brains of both transgenic mice and human patients with .beta.-amyloid pathol. The authors collected sera from patients with **Alzheimer** disease who received a primary injection of pre-aggregated A.beta.42 followed by one booster injection in a placebo-**controlled** study. Antibodies in immune sera recognized .beta.-amyloid plaques, diffuse A.beta. deposits, and vascular .beta.-amyloid in brain blood vessels. The antibodies did not cross-react with native full-length .beta.-amyloid precursor protein or its physiol. derivs., including sol. A.beta.42. Thus, **vaccination** of AD patients with A.beta.42 induces antibodies that have a high degree of selectivity for the pathogenic target structures. Whether **vaccination** to produce antibodies against .beta.-amyloid will halt the cognitive

decline in AD will depend upon clin. assessments over time.

IT **107761-42-2**, Glycopeptide (human clone 9-110 **amyloid** A4 peptide moiety)
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(pre-aggregated; antibodies formation to .beta.-**amyloid** by **vaccination** of **Alzheimer**'s **disease** patients with **amyloid** .beta.42 peptide)

REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 4 OF 29 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:555371 HCAPLUS
DOCUMENT NUMBER: 137:139348
TITLE: Molecular antigen array for **vaccines** against infectious disease, cancer, allergies and autoimmune diseases
INVENTOR(S): Maurer, Patrick; Lechner, Franziska; Ortmann, Rainer; Lueoend, Rainer; Staufenbiel, Matthias; Frey, Peter; Renner, Wolfgang A.; Bachmann, Martin; Tissot, Alain; Sebbel, Peter; Piossek, Christine
PATENT ASSIGNEE(S): Cytos Biotechnology A.-G., Switz.; Novartis Pharma A.-G.
SOURCE: PCT Int. Appl., 418 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002056907	A2	20020725	WO 2002-IB168	20020121

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:
US 2001-262379P P 20010119
US 2001-288549P P 20010504
US 2001-326998P P 20011005
US 2001-331045P P 20011107

AB The present invention is related to the fields of mol. biol., virol., immunol. and medicine. The invention provides a compn. comprising an ordered and repetitive antigen or antigenic determinant array. The invention also provides a process for producing an antigen or antigenic determinant in an ordered and repetitive array. The ordered and repetitive antigen or antigenic determinant is useful in the prodn. of **vaccines** for the **treatment** of infectious diseases, the **treatment** of

allergies and as a pharmaccine to **prevent** or cure cancer and to efficiently induce self-specific immune responses, in particular antibody responses.

IT **107761-42-2**, **Amyloid** .beta. 1-42
444309-74-4
RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(mol. antigen array for **vaccines** against infectious **disease**, cancer, allergies and autoimmune **diseases**)

IT **444137-68-2 444137-69-3 444137-70-6**
RL: PRP (Properties)
(unclaimed protein sequence; mol. antigen array for **vaccines** against infectious disease, cancer, allergies and autoimmune diseases)

L8 ANSWER 5 OF 29 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:540135 HCAPLUS
DOCUMENT NUMBER: 137:108295
TITLE: **Vaccines** comprising all-D fibril peptides for **prevention** and **treatment** of **Alzheimer**'s and **amyloid**-related **diseases**
INVENTOR(S): Chalifour, Robert; Hebert, Lise; Kong, Xianqi; Gervais, Francine
PATENT ASSIGNEE(S): Can.
SOURCE: U.S. Pat. Appl. Publ., 28 pp., Cont.-in-part of U.S. Ser. No. 724,842.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002094335	A1	20020718	US 2001-867847	20010529
WO 2002096937	A2	20021205	WO 2002-CA763	20020529

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 1999-168594P P 19991129
US 2000-724842 A2 20001128
US 2001-867847 A 20010529

AB The present invention relates to a stereochem. based "non-self" antigen **vaccine** for the **prevention** and/or **treatment** of **Alzheimer**'s and other **amyloid** related **diseases**. The present invention provides a **vaccine** for the **prevention** and **treatment** of **Alzheimer**'s and other **amyloid** related

diseases, which overcomes the drawbacks assocd. with using naturally occurring peptides, proteins or immunogens. The **vaccine** comprises fibril peptides consisting of all- D-amino acids.

IT **342877-52-5 342877-55-8 342877-58-1**
342877-61-6 342877-63-8 342877-66-1
342877-69-4 342877-71-8 342877-73-0
342877-74-1 342877-75-2 342877-97-8
342878-00-6 342878-03-9 342878-06-2
342878-09-5 442915-40-4
RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**vaccines** comprising all-D fibril peptides for **prevention** and **treatment** of **Alzheimer**'s and **amyloid**-related **diseases**)

L8 ANSWER 6 OF 29 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:475976 HCAPLUS
DOCUMENT NUMBER: 137:383644
TITLE: Patients with **Alzheimer** **disease** have lower levels of serum anti-**amyloid** peptide antibodies than healthy elderly individuals
AUTHOR(S): Weksler, Marc E.; Relkin, Norman; Turkenich, Rimma; LaRusse, Susan; Zhou, Ling; Szabo, Paul
CORPORATE SOURCE: Department of Medicine, Weill Medical College of Cornell University, New York, NY, 10021, USA
SOURCE: Experimental Gerontology (2002), 37(7), 943-948
CODEN: EXGEAB; ISSN: 0531-5565
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Active **immunization** with the human amyloid peptide (A.beta.42) or passive **immunization** with anti-A.beta.42 antibodies protects mice that express a mutant human amyloid precursor protein (APP) transgene from cerebral amyloid deposits. If anti-A.beta.42 antibodies protect APP-transgenic mice, a model of **Alzheimer**'s disease (**AD**), a high titer of anti-A.beta.42 antibodies may protect humans from **AD**. The titer of anti-A.beta.42 antibodies in serum from individuals with and without late onset AD was measured using an ELISA. The titer of Ig (IgM, IgG and IgA) and IgG anti-A.beta.42 peptide antibodies was significantly higher in serum from elderly **controls** than AD patients. Furthermore, IgG but not Ig anti-A.beta.42 antibodies distinguished sera from AD patients and elderly **controls** that did not have the apolipoprotein E4 allele. The low titer of anti-A.beta.42 antibodies in AD patients does not reflect the well-established, age-assocd. defect in the antibody response to most protein antigens, as there was no pos. correlation between the serum titer of anti-A.beta.42 antibodies and anti-influenza hemagglutinin antibodies induced by influenza **vaccine** in elderly humans. The lower titer of serum anti-A.beta.42 peptide antibodies in AD patients may reflect the reported specific impairment of helper T cell activity for B cells that produce anti-amyloid-.beta.42 peptide antibodies in APP-transgenic mice.

IT **107761-42-2**, Glycopeptide (human clone 9-110 **amyloid** A4 peptide moiety)
RL: BSU (Biological study, unclassified); BIOL (Biological study)

(patients with **Alzheimer disease** have lower levels of serum anti-**amyloid** peptide antibodies than healthy elderly individuals)

REFERENCE COUNT: 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 7 OF 29 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:353475 HCAPLUS
DOCUMENT NUMBER: 136:363863
TITLE: Peptides for use in the **treatment** of **Alzheimer**'s disease
INVENTOR(S): Milton, Nathaniel Gavin Nicolas
PATENT ASSIGNEE(S): Insight Biotechnology Limited, UK
SOURCE: PCT Int. Appl., 44 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002036614	A2	20020510	WO 2001-GB4843	20011101
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002012471	A5	20020515	AU 2002-12471	20011101

PRIORITY APPLN. INFO.:
GB 2000-26738 A 20001101
GB 2000-26739 A 20001101
WO 2001-GB4843 W 20011101

AB Antisense peptides that correspond to Amyloid-.beta. protein residues 1-43 are identified, and are used to identify protein binding sites on enzymes that interact with Amyloid-.beta.. The antisense peptides can be used as, or to identify, **therapeutic** agents that **prevent Amyloid** -.beta. cytotoxicity, and may be useful in the **treatment** of **Alzheimer**'s **disease**. The antisense peptides show sequence similarity to the protein kinase cdc2, and it has now been found that the cytotoxic form of A.beta. is phosphorylated.

IT **422613-26-1**
RL: BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(peptides for use in the **treatment** of **Alzheimer**'s disease)

L8 ANSWER 8 OF 29 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:332668 HCAPLUS
DOCUMENT NUMBER: 136:345817

TITLE: Methods and compositions for the **treatment** and/or diagnosis of neurological diseases and disorders
INVENTOR(S): Solomon, Beka; Frenkel, Dan
PATENT ASSIGNEE(S): Israel
SOURCE: U.S. Pat. Appl. Publ., 75 pp., Cont.-in-part of U.S. Ser. No. 629,971.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002052311	A1	20020502	US 2001-808037	20010315
WO 2002074243	A2	20020926	WO 2002-US8042	20020315

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:
US 1999-152417P P 19990903
US 1999-473653 A2 19991229
US 2000-629971 A2 20000731
US 2001-808037 A1 20010315

AB A method of **immunizing** against plaque-forming diseases using display technol. is provided. The method utilize novel agents, or pharmaceutical compns. for **vaccination** against plaque-forming diseases which rely upon presentation of an antigen or epitope on a display vehicle. The method further includes agents, or pharmaceutical compns. for **vaccination** against plaque-forming diseases, which rely upon presentation of an antibody, or an active portion thereof, on a display vehicle. Whether antigens or antibodies are employed, disaggregation of plaques results from the **immunization**. The methods of the present invention also generally relates to **treating** and/or diagnosing neurol. diseases and disorders of the central nervous, regardless of whether the disease or disorder is plaque-forming or non-plaque forming.

IT **419018-23-8**
RL: PRP (Properties)
(unclaimed protein sequence; methods and compns. for **treatment** and/or diagnosis of neurol. diseases and disorders)

L8 ANSWER 9 OF 29 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:332217 HCAPLUS
DOCUMENT NUMBER: 136:339487
TITLE: Fusion proteins comprising .beta.-**amyloid** peptide and heat shock protein for **immunization treatments**

of **Alzheimer's disease**

INVENTOR(S): Ghirardi, Silvia; Armani, Elisabetta; Amari, Gabriele; Puccini, Paola; Imbimbo, Bruno; Villetti, Gino

PATENT ASSIGNEE(S): Chiesi Farmaceutici S.P.A., Italy; Ghirardi Silvia

SOURCE: PCT Int. Appl., 33 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002034777	A1	20020502	WO 2001-EP12242	20011023

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2002023640	A5	20020506	AU 2002-23640	20011023

PRIORITY APPLN. INFO.: IT 2000-MI2299 A 20001024
WO 2001-EP12242 W 20011023

AB The present invention is related to fusion proteins (A.beta.-Hsp) (III) and their use in the **treatment** or prophylaxis of **disorders** assocd. with an accumulation of .beta.-**amyloid**, specifically in patients with **Alzheimer's disease**. Said fusion proteins are derived from the condensation of .beta.-amyloid protein or fragments thereof (A.beta.) with a heat shock protein (Hsp). The .beta.-amyloid peptide is human .beta.-amyloid peptide (1-39), (1-40) or (1-42); and the heat shock protein is Hsp25, Hsp27, Hsp28, Hsp60, Hsp70 or Hsp90.

IT **419018-03-4P**
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(amino acid sequence; fusion proteins comprising .beta.-**amyloid** peptide and heat shock protein for **immunization treatments** of **Alzheimer's disease**)

IT **107761-42-2D**, Human .beta.-**amyloid** peptide(1-42), heat shock protein conjugate **131438-79-4D**, Human .beta.-**amyloid** peptide(1-40), heat shock protein conjugate
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(fusion proteins comprising .beta.-**amyloid** peptide and heat shock protein for **immunization treatments** of **Alzheimer's disease**)

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN

THE RE FORMAT

L8 ANSWER 10 OF 29 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:318788 HCAPLUS
DOCUMENT NUMBER: 137:4739
TITLE: **Immunization** reverses memory deficits without reducing brain A.beta. burden in **Alzheimer**'s disease model
AUTHOR(S): Dodart, J. C.; Bales, K. R.; Gannon, K. S.; Greene, S. J.; DeMattos, R. B.; Mathis, C.; DeLong, C. A.; Wu, S.; Wu, X.; Holtzman, D. M.; Paul, S. M.
CORPORATE SOURCE: Neuroscience Discovery Research, Lilly Corporate Center, Lilly Research Laboratories, Indianapolis, IN, 46285, USA
SOURCE: Nature Neuroscience (2002), 5(5), 452-457
CODEN: NANEFN; ISSN: 1097-6256
PUBLISHER: Nature America Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The authors have previously shown that chronic **treatment** with the monoclonal antibody m266, which is specific for **amyloid** .beta.-peptide (A.beta.), increases plasma concns. of A.beta. and reduces A.beta. burden in the PDAPP transgenic mouse model of **Alzheimer**'s **disease** (**AD**). The authors now report that administration of m266 to PDAPP mice can rapidly reverse memory deficits in both an object recognition task and a hole board learning and memory task, but without altering brain A.beta. burden. The authors also found that an A.beta./antibody complex was present in both the plasma and the cerebrospinal fluid of m266-**treated** mice. Our data indicate that passive **immunization** with this anti-A.beta. monoclonal antibody can very rapidly reverse memory impairment in certain learning and memory tasks in the PDAPP mouse model of AD, owing perhaps to enhanced peripheral clearance and (or) sequestration of a sol. brain A.beta. species.
IT **107761-42-2D**, Glycopeptide (human clone 9-110 **amyloid** A4 peptide moiety), immune complexes-contg.
131438-79-4D, immune complexes-contg.
RL: BSU (Biological study, unclassified); BIOL (Biological study) (of serum and cerebrospinal fluid in passive **immunization** in **Alzheimer**'s **disease** model)
REFERENCE COUNT: 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 11 OF 29 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:51195 HCAPLUS
DOCUMENT NUMBER: 136:112669
TITLE: **Prevention** and **treatment** of **Alzheimer**'s disease
INVENTOR(S): Lannfelt, Lars; Naeslund, Jan; Westlind-Danielsson, Anita; Nilsberth, Camilla
PATENT ASSIGNEE(S): Swed.
SOURCE: PCT Int. Appl., 25 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002003911	A2	20020117	WO 2001-SE1553	20010704
WO 2002003911	A3	20020411		
WO 2002003911	C1	20020620		

W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

AU 2001068005	A5	20020121	AU 2001-68005	20010705
US 2002162129	A1	20021031	US 2001-899815	20010709

PRIORITY APPLN. INFO.:

EP 2000-202387	A	20000707
US 2000-217098P	P	20000710
WO 2001-SE1553	W	20010704

AB The present invention relates to **prevention** and **treatment** of **Alzheimer**'s disease (**AD**). More specifically, the invention relates to use of a non-wild type protofibril or compd.(s) with protofibril forming activity for active **immunization** in the purpose of **treating** or **preventing** AD. The invention further relates to a peptide, A.beta.-Arc, with high protofibril forming activity as well as several applications thereof, such as antibodies against said peptide for passive **immunization** against AD.

IT **389151-08-0**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**prevention** and **treatment** of **Alzheimer**'s disease)

IT **159647-22-0**
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**prevention** and **treatment** of **Alzheimer**'s disease)

L8 ANSWER 12 OF 29 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:43680 HCAPLUS
DOCUMENT NUMBER: 136:400445
TITLE: Number of A.beta. inoculations in APP+PS1 transgenic mice influences antibody titers, microglial activation, and congophilic plaque levels
AUTHOR(S): Wilcock, Donna M.; Gordon, Marcia N.; Ugen, Kenneth E.; Gottschall, Paul E.; Dicarlo, Giovanni; Dickey, Chad; Boyett, Kristal W.; Jantzen, Paul T.; Connor, Karen E.; Melachrino, Jason; Hardy, John; Morgan, David
CORPORATE SOURCE: Alzheimer's Research Laboratory, Department of Pharmacology, University of South Florida, Tampa, FL, USA

SOURCE: DNA and Cell Biology (2001), 20(11), 731-736
CODEN: DCEBE8; ISSN: 1044-5498
PUBLISHER: Mary Ann Liebert, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB There have been several reports on the use of .beta.-**amyloid** (A.beta.) **vaccination** in different mouse models of **Alzheimer's disease** (**AD**) and its effects on pathol. and cognitive function. In this report, the histopathol. findings in the APP+PS1 doubly transgenic mouse were compared after three, five, or nine A.beta. inoculations. The no. of inoculations influenced the effects of **vaccination** on Congo red levels, microglia activation, and anti-A.beta. antibody titers. After three inoculations, the antibody titer of transgenic mice was substantially lower than that found in nontransgenic animals. However, after nine inoculations, the levels were considerably higher in both genotypes and no longer distinguishable statistically. The no. of inoculations influenced CD45 expression, an indicator of microglial activation. There was an initial upregulation, which was significant after five inoculations, but by nine inoculations, the extent of microglial activation was equiv. to that in mice given **control vaccinations**. Along with this increased CD45 expression, there was a correlative redn. in staining by Congo red, which stains compact plaques. When data from the mice from all groups were combined, there was a significant correlation between activation of microglia and Congo red levels, suggesting that microglia play a role in the clearance of compact plaque.

IT **107761-42-2**, Amyloid .beta. 1-42
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(no. of A.beta.1-42 inoculations in transgenic mice carrying amyloid precursor protein and presenilin 1 transgenes influences antibody titers, microglial activation, and congophilic plaque levels)

REFERENCE COUNT: 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 13 OF 29 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:10296 HCAPLUS
DOCUMENT NUMBER: 136:68700
TITLE: Tyrosine cross-linked oligomers of amyloid peptide: Pathology and immunotherapy
INVENTOR(S): Bush, Ashley; Cherny, Robert
PATENT ASSIGNEE(S): Prana Biotechnology Limited, Australia; The General Hospital Corporation
SOURCE: PCT Int. Appl., 59 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002000245	A1	20020103	WO 2001-AU786	20010628

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD,

GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

AU 2001068828 A5 20020108 AU 2001-68828 20010628
PRIORITY APPLN. INFO.: US 2000-214779P P 20000628
US 2000-242177P P 20001023
WO 2001-AU786 W 20010628

AB This invention relates to methods and compns. for the **treatment** or alleviation of **Alzheimer**'s disease and of other conditions related to abnormal protein aggregation. In particular, the invention relates to methods and compns. for the immunotherapy of **Alzheimer**'s disease, Parkinson's disease, and cataract. In one aspect the invention provides a method of prophylaxis, **treatment** or alleviation of a condition characterized by pathol. aggregation and accumulation of a specific protein assocd. with oxidative damage and formation of tyrosine cross-links, comprising the step of **immunizing** a subject in need of such **treatment** with an **immunizing**-ED of one or more tyrosine cross-linked compds., and optionally also comprising copper ions complexed to the compd. Alternatively passive **immunization** against a tyrosine cross-linked compd. may be used.

IT **107761-42-2D**, Glycopeptide (human clone 9-110 **amyloid** A4 peptide moiety), tyrosine cross-linked oligomers
131438-79-4D, tyrosine cross-linked oligomers
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(immunotherapy of **Alzheimer**'s **disease** with)

REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 14 OF 29 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:827658 HCAPLUS
DOCUMENT NUMBER: 136:400139
TITLE: Reduced effectiveness of A.beta.1-42 **immunization** in APP transgenic mice with significant amyloid deposition
AUTHOR(S): Das, Pritam; Murphy, M. Paul.; Younkin, Linda H.; Younkin, Steven. G.; Golde, Todd E.
CORPORATE SOURCE: Department of Neurosciences, Mayo Clinic Jacksonville, Jacksonville, FL, 32224, USA
SOURCE: Neurobiology of Aging (2001), 22(5), 721-727
CODEN: NEAGDO; ISSN: 0197-4580
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB **Vaccinations** with A.beta.1-42 have been shown to reduce **amyloid** burden in transgenic models of **Alzheimer**'s **disease** (**AD**). The authors have further tested the efficacy of A.beta.1-42 **immunization** in the Tg2576 mouse model of AD by **immunizing** one group of mice with minimal A.beta. deposition, one group of mice with modest A.beta.

deposition, and one group with significant A.beta. deposition. The effects of **immunization** on A.beta. deposition were examd. using biochem. and immunohistochem. methods. In Tg2576 mice **immunized** prior to significant amyloid deposition, A.beta.1-42 **immunization** was highly effective. Biochem. extd. A.beta.40 and A.beta.42 levels were significantly reduced and immunohistochem. plaque load was also reduced. **Immunization** of mice with modest amts. of pre-existing A.beta. deposits selectively reduced A.beta.42 without altering A.beta.40, although plaque load was reduced. In contrast, in Tg2576 mice with significant pre-existing A.beta. loads, A.beta.1-42 **immunization** only minimally decreased A.beta.42 levels, whereas no alteration in A.beta.40 levels or in plaque load was obsd. These results indicate that in Tg2576 mice, A.beta.1-42 **immunization** is more effective at **preventing** addnl. A.beta. accumulation and does not result in significant clearance of pre-existing A.beta. deposits.

IT **131438-79-4**
RL: ADV (Adverse effect, including toxicity); BSU (Biological study, unclassified); BIOL (Biological study)
(reduced effectiveness of A.beta.1-42 **immunization** in APP transgenic mice with significant amyloid deposition)

IT **107761-42-2**, Glycopeptide (human clone 9-110 amyloid A4 peptide moiety)
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(reduced effectiveness of A.beta.1-42 **immunization** in APP transgenic mice with significant amyloid deposition)

REFERENCE COUNT: 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 15 OF 29 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:661132 HCAPLUS
DOCUMENT NUMBER: 136:261466
TITLE: **Immunization** with a nontoxic/nonfibrillar **amyloid**-.beta. homologous peptide reduces **Alzheimer**'s **disease**-associated pathology in transgenic mice
AUTHOR(S): Sigurdsson, Einar M.; Scholtzova, Henrieta; Mehta, Pankaj D.; Frangione, Blas; Wisniewski, Thomas
CORPORATE SOURCE: Department of Neurology, New York University School of Medicine, New York, NY, 10016, USA
SOURCE: American Journal of Pathology (2001), 159(2), 439-447
CODEN: AJPAA4; ISSN: 0002-9440
PUBLISHER: American Society for Investigative Pathology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Transgenic mice with brain amyloid-.beta. (A.beta.) plaques **immunized** with aggregated A.beta.1-42 have reduced cerebral amyloid burden. However, the use of A.beta.1-42 in humans may not be appropriate because it crosses the blood brain barrier, forms toxic fibrils, and can seed fibril formation. We report that **immunization** in transgenic APP mice (Tg2576) for 7 mo with a sol. nonamyloidogenic, nontoxic A.beta. homologous peptide reduced

cortical and hippocampal brain amyloid burden by 89% and 81%, resp. Concurrently, brain levels of sol. A.beta.1-42 were reduced by 57%. Ramified microglia expressing interleukin-1.beta. assocd. with the A.beta. plaques were absent in the **immunized** mice indicating reduced inflammation in these animals. These promising findings suggest that **immunization** with nonamyloidogenic A.beta. derivs. represents a potentially safer **therapeutic** approach to reduce **amyloid** burden in **Alzheimer**'s **disease**, instead of using toxic A.beta. fibrils.

IT **107761-42-2**, .beta.-**Amyloid** 1-42
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**immunization** with a nontoxic/nonfibrillar **amyloid**-.beta. homologous peptide reduces **Alzheimer**'s **disease**)

REFERENCE COUNT: 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 16 OF 29 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:590755 HCAPLUS
DOCUMENT NUMBER: 135:342776
TITLE: A.beta. **immunization**: moving A.beta. peptide from brain to blood
AUTHOR(S): Lee, Virginia M.-Y.
CORPORATE SOURCE: Center for Neurodegenerative Disease Research, Department of Pathology and Laboratory Medicine, University of Pennsylvania School of Medicine, Philadelphia, PA, 19104, USA
SOURCE: Proceedings of the National Academy of Sciences of the United States of America (2001), 98(16), 8931-8932
CODEN: PNASA6; ISSN: 0027-8424
PUBLISHER: National Academy of Sciences
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review, with refs., discussing **amyloid** .beta. (A.beta.), the major component of senile plaques, as a realistic target for developing effective **therapies** for **Alzheimer**'s **disease** (**AD**). The study conducted by DeMattos et al., who provide mechanistic insights on an approach that can lead to the elimination of amyloid deposits in the brains of transgenic mice that develop AD amyloidosis, and the work of Bard et al. are also discussed. Examples of a process for plaque turnover in a transgenic mouse model of amyloidosis are described.

IT **107761-42-2**, Glycopeptide (human clone 9-110 **amyloid** A4 peptide moiety)
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**immunization** with **amyloid** peptide for **treatment** of **Alzheimer**'s **disease**)

REFERENCE COUNT: 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 17 OF 29 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:545521 HCAPLUS
DOCUMENT NUMBER: 135:136411

TITLE: Heat shock/stress protein complexes as **vaccines** against neurodegenerative disorders
INVENTOR(S): Srivastava, Pramod K.
PATENT ASSIGNEE(S): University of Connecticut Health Center, USA
SOURCE: PCT Int. Appl., 65 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001052890	A1	20010726	WO 2001-US1825	20010118

W: AU, CA, JP
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR
PRIORITY APPLN. INFO.: US 2000-489216 A 20000121
AB The present invention relates to pharmaceutical compns. comprising complexes of heat shock proteins (hsps) in assocn. with antigenic mols. for use in **treatment** and **prevention** of neurodegenerative disorders and disease. The invention further relates to methods for the use of such pharmaceutical compns. as immunotherapeutic agents for the **treatment** and **prevention** of neurodegenerative disorders and disease.
IT **107761-42-2**, Glycopeptide (human clone 9-110 **amyloid** A4 peptide moiety) **131438-79-4**
RL: PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(heat shock/stress protein-.beta. **amyloid** complexes as **vaccines** against neurodegenerative **disorders**)
REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 18 OF 29 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:455699 HCAPLUS
DOCUMENT NUMBER: 135:193890
TITLE: Early-onset amyloid deposition and cognitive deficits in transgenic mice expressing a double mutant form of amyloid precursor protein 695
AUTHOR(S): Chishti, M. Azhar; Yang, Dun-Shen; Janus, Christopher; Phinney, Amie L.; Horne, Patrick; Pearson, Jacqueline; Strome, Robert; Zuker, Noah; Loukides, James; French, Janet; Turner, Sherry; Lozza, Gianluca; Grilli, Mariagrazia; Kunicki, Suzanne; Morissette, Celine; Paquette, Julie; Gervais, Francine; Bergeron, Catherine; Fraser, Paul E.; Carlson, George A.; St. George-Hyslop, Peter; Westaway, David
CORPORATE SOURCE: Centre for Research in Neurodegenerative Diseases, University of Toronto, Toronto, ON, M5S 3H2, Can.
SOURCE: Journal of Biological Chemistry (2001), 276(24), 21562-21570
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular

Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB We have created early-onset transgenic (Tg) models by exploiting the synergistic effects of familial **Alzheimer**'s **disease** mutations on **amyloid** .beta.-peptide (A.beta.) biogenesis. TgCRND8 mice encode a double mutant form of amyloid precursor protein 695 (KM670/671NL+V717F) under the **control** of the PrP gene promoter. Thioflavine S-pos. A.beta. amyloid deposits are present at 3 mo, with dense-cored plaques and neuritic pathol. evident from 5 mo of age. TgCRND8 mice exhibit 3,200-4,600 pmol of A.beta.42 per g brain at age 6 mo, with an excess of A.beta.42 over A.beta.40. High level prodn. of the pathogenic A.beta.42 form of A.beta. peptide was assocd. with an early impairment in TgCRND8 mice in acquisition and learning reversal in the ref. memory version of the Morris water maze, present by 3 mo of age. Notably, learning impairment in young mice was offset by **immunization** against A.beta.42. **Amyloid** deposition in TgCRND8 mice was enhanced by the expression of presenilin 1 transgenes including familial **Alzheimer**'s **disease** mutations; for mice also expressing a M146L+L286V presenilin 1 transgene, **amyloid** deposits were apparent by 1 mo of age. The Tg mice described here suggest a potential to investigate aspects of **Alzheimer**'s disease pathogenesis, prophylaxis, and **therapy** within short time frames.

IT **107761-42-2**, Glycopeptide (human clone 9-110 amyloid A4 peptide moiety) **131438-79-4**, Human .beta.-amyloid peptide(1-40)
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(early-onset amyloid deposition and cognitive deficits in transgenic mice expressing a double mutant form of amyloid precursor protein 695)

REFERENCE COUNT: 73 THERE ARE 73 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 19 OF 29 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:416788 HCAPLUS
DOCUMENT NUMBER: 135:18553
TITLE: **Vaccine** for the **prevention** and **treatment** of **Alzheimer**'s and **amyloid** related **diseases**
INVENTOR(S): Chalifour, Robert; Hebert, Lise; Kong, Xianqi; Gervais, Francine
PATENT ASSIGNEE(S): Neurochem Inc., Can.
SOURCE: PCT Int. Appl., 31 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001039796	A2	20010607	WO 2000-CA1413	20001129

WO 2001039796 A3 20011206
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
BR 2000016022 A 20020806 BR 2000-16022 20001129
EP 1235587 A2 20020904 EP 2000-981111 20001129
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
NO 2002002531 A 20020712 NO 2002-2531 20020528
PRIORITY APPLN. INFO.: US 1999-168594P P 19991129
US 2000-724842 A 20001128
WO 2000-CA1413 W 20001129

AB The present invention relates to a stereochem. based "non-self" antigen **vaccine** for the **prevention** and/or **treatment** of **Alzheimer**'s and other **amyloid** related **diseases**. The present invention provides a **vaccine** for the **prevention** and **treatment** of **Alzheimer**'s and other **amyloid** related **diseases**, which overcomes the drawbacks assocd. with using naturally occurring peptides, proteins or immunogens.

IT **226707-64-8P 342877-52-5P 342877-55-8P**
342877-58-1P 342877-61-6P 342877-63-8P
342877-66-1P 342877-69-4P 342877-71-8P
342877-73-0P 342877-74-1P 342877-75-2P
342877-97-8P 342878-00-6P 342878-03-9P
342878-06-2P 342878-09-5P 342896-25-7P
342896-48-4P
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(**vaccine** for **prevention** and **treatment** of **Alzheimer**'s and **amyloid** related **diseases** using all-D peptides that elicit immune response to **amyloid** protein)

L8 ANSWER 20 OF 29 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:185883 HCAPLUS
DOCUMENT NUMBER: 134:236224
TITLE: Agents and compositions and methods utilizing same useful in diagnosing and/or **treating** or **preventing** plaque forming diseases
INVENTOR(S): Solomon, Beka; Frenkel, Dan; Hanan, Eilat
PATENT ASSIGNEE(S): Ramot University Authority for Applied Research & Industrial Development, Israel
SOURCE: PCT Int. Appl., 120 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English

FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001018169	A2	20010315	WO 2000-IL518	20000831
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1180938	A2	20020227	EP 2000-954883	20000831
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, MC, IE, SI, LT, LV, FI, RO				

PRIORITY APPLN. INFO.:

US 1999-152417P	P	19990903
US 1999-473653	A	19991229
US 2000-629971	A	20000731
WO 2000-IL518	W	20000831

9-3-99 *
12-29-99

AB A method of **immunizing** against plaque forming diseases using display technol. is provided. The method utilizes novel agents, or pharmaceutical compns. for **vaccination** against plaque forming diseases which rely upon presentation of an antigen or epitope on a display vehicle. The method further includes agents, or pharmaceutical compns. for **vaccination** against plaque forming diseases, which rely upon presentation of an antibody, or an active portion thereof, on a display vehicle. Whether antigens or antibodies are employed, disaggregation of plaques results from the **immunization**.

IT **134500-80-4P**
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(display vehicles encoding human PrP epitope or anti-.beta. **amyloid** antibody scFv for diagnosing and/or **treating** or **preventing** plaque forming **diseases**)

L8 ANSWER 21 OF 29 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2000:900782 HCAPLUS
DOCUMENT NUMBER: 134:55503
TITLE: Immunological **control** of .beta.-amyloid levels in vivo
INVENTOR(S): Raso, Victor
PATENT ASSIGNEE(S): Boston Biomedical Research Institute, USA
SOURCE: PCT Int. Appl., 82 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

WO 2000077178 A1 20001221 WO 2000-US16551 20000615
W: AU, CA, JP
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
US 2002102261 A1 20020801 US 2001-992800 20011106
US 2002136718 A1 20020926 US 2001-992994 20011106
PRIORITY APPLN. INFO.: US 1999-139408P P 19990616
US 2000-594366 A3 20000615

AB The present invention provides an antibody which catalyzes hydrolysis of .beta.-amyloid at a predetd. amide linkage. The antibody preferentially binds a transition state analog which mimics the transition state adopted by .beta.-amyloid during hydrolysis at a predetd. amide linkage. Specific antibodies provided include those which catalyze the hydrolysis at the amyloid linkages between residues 39 and 40, 40 and 41, and 41 and 42 of .beta.-amyloid. The present antibody also provides a vectorized antibody which is characterized by the ability to cross the blood brain barrier and also catalyze the hydrolysis of .beta.-amyloid. Also provided are methods for sequestering free .beta.-amyloid in the blood stream, for reducing levels of .beta.-amyloid in the brain, for reducing the level of circulating .beta.-amyloid, for **preventing** the formation of amyloid plaques in the rain and for disaggregating amyloid plaques. Finally, this invention also provides a method of generating antibodies by **immunizing** an animal with antigen comprised of an epitope which has a statine analog or which utilizes reduced peptide bond analogs to mimic the conformation of a hydrolysis transition state of a polypeptide.

IT **134500-80-4**
RL: BSU (Biological study, unclassified); PRP (Properties); REM (Removal or disposal); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(antibodies for catalyzing hydrolysis of .beta.-amyloid and reducing .beta.-amyloid in brain)

IT **313474-75-8**
RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(antibodies for catalyzing hydrolysis of .beta.-amyloid and reducing .beta.-amyloid in brain)

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 22 OF 29 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2000:861516 HCAPLUS
DOCUMENT NUMBER: 134:28431
TITLE: **Prevention** and **treatment** of amyloidogenic disease
INVENTOR(S): Schenk, Dale B.
PATENT ASSIGNEE(S): Neuralab Limited, Bermuda
SOURCE: PCT Int. Appl., 140 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

WO 2000072876 A2 20001207 WO 2000-US15239 20000601
WO 2000072876 A3 20010503
W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
EP 1185296 A2 20020313 EP 2000-938075 20000601
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO
BR 2000011103 A 20020319 BR 2000-11103 20000601
NO 2001005758 A 20020130 NO 2001-5758 20011126
PRIORITY APPLN. INFO.: US 1999-137010P P 19990601
WO 2000-US15239 W 20000601

6-1999

AB The authors discloses methods for immunotherapy of **amyloid diseases**, including **Alzheimer's disease**, prion **diseases**, and familial **amyloid** neuropathies. In one example, **Alzheimer's disease** -prone mice were **immunized** with **amyloid** peptide (A.beta.1-42). In contrast to **control** mice, **treated** mice exhibited a lack of amyloid plaques, neuritic pathol., and astrocytosis. In a second example, **Alzheimer's disease**-prone mice were passively **immunized** with antibodies to **amyloid** peptides. **Treated** mice exhibited a significant decrease in cerebral A.beta. levels and a redn. in amyloid load.

IT **107761-42-2**, Glycopeptide (human clone 9-110 **amyloid** A4 peptide moiety) **312263-74-4**
RL: PRP (Properties)
(unclaimed sequence; **prevention** and **treatment** of amyloidogenic **disease**)

L8 ANSWER 23 OF 29 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2000:552560 HCAPLUS
DOCUMENT NUMBER: 133:236580
TITLE: Peripherally administered antibodies against **amyloid** .beta.-peptide enter the central nervous system and reduce pathology in a mouse model of **Alzheimer disease**
AUTHOR(S): Bard, Frederique; Cannon, Catherine; Barbour, Robin; Burke, Rae-Lyn; Games, Dora; Grajeda, Henry; Guido, Teresa; Hu, Kang; Huang, Jiping; Johnson-Wood, Kelly; Khan, Karen; Kholodenko, Dora; Lee, Mike; Lieberburg, Ivan; Motter, Ruth; Nguyen, Minh; Soriano, Ferdie; Vasquez, Nicki; Weiss, Kim; Welch, Brent; Seubert, Peter; Schenk, Dale; Yednock, Ted
CORPORATE SOURCE: Elan Pharmaceuticals, South San Francisco, CA, 94080, USA
SOURCE: Nature Medicine (New York) (2000), 6(8), 916-919
CODEN: NAMEFI; ISSN: 1078-8956
PUBLISHER: Nature America Inc.
DOCUMENT TYPE: Journal

LANGUAGE: English

AB One hallmark of **Alzheimer disease** is the accumulation of **amyloid** .beta.-peptide in the brain and its deposition as plaques. Mice transgenic for an **amyloid** .beta. precursor protein (APP) mini-gene driven by a platelet-derived (PD) growth factor promoter (PDAPP mice), which overexpress one of the **disease**-linked mutant forms of the human **amyloid** precursor protein, show many of the pathol. features of **Alzheimer disease**, including extensive deposition of extracellular **amyloid** plaques, astrocytosis and neuritic dystrophy. Active **immunization** of PDAPP mice with human amyloid .beta.-peptide reduces plaque burden and its assocd. pathologies. Several hypotheses have been proposed regarding the mechanism of this response. Here the authors report that peripheral administration of antibodies against amyloid .beta.-peptide, was sufficient to reduce amyloid burden. Despite their relatively modest serum levels, the passively administered antibodies were able to enter the central nervous system, decorate plaques and induce clearance of preexisting amyloid. When examd. in an ex vivo assay with sections of PDAPP or **Alzheimer disease** brain tissue, antibodies against **amyloid** .beta.-peptide triggered microglial cells to clear plaques through Fc receptor-mediated phagocytosis and subsequent peptide degrdn. These results indicate that antibodies can cross the blood-brain barrier to act directly in the central nervous system and should be considered as a **therapeutic** approach for the **treatment** of **Alzheimer** disease and other neurol. disorders.

IT **107761-42-2**, Glycopeptide (human clone 9-110 **amyloid** A4 peptide moiety)
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(plaque clearance in **Alzheimer**'s **disease** model is promoted by antibodies to)

REFERENCE COUNT: 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 24 OF 29 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2000:513524 HCAPLUS
DOCUMENT NUMBER: 133:129886
TITLE: Application of a viral complement-inhibitory protein in the **treatment** and diagnosis of **Alzheimer**'s disease
INVENTOR(S): Kotwal, Girish J.; Daly, James, IV
PATENT ASSIGNEE(S): University of Louisville Research Foundation, Inc., USA
SOURCE: PCT Int. Appl., 96 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000043027	A1	20000727	WO 2000-US1115	20000119
W: AU, CA, JP, US				

RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
PRIORITY APPLN. INFO.: US 1999-116328P P 19990119
AB The present invention provides a novel **treatment** for senile dementia (**Alzheimer**'s Type), comprising administering an anti-complement protein to a patient in need of such **treatment** in an amt. sufficient to inhibit the complement cascade and thereby inhibit the prodn. or enlargement of amyloid plaques in the brain of the patient. The present invention further provides pharmaceutical compns. comprising anti-complement protein, or derivs. thereof, and/or pharmaceutically acceptable salts thereof in a variety of unique pharmaceutical dosage forms.
IT **134548-35-9**, 652-751-Glycoprotein (human clone .lambda.APCP168i4 **amyloid** A4 precursor protein moiety reduced)
RL: PRP (Properties)
(unclaimed protein sequence; application of a viral complement-inhibitory protein in the **treatment** and diagnosis of **Alzheimer**'s **disease**)
REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 25 OF 29 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1999:466239 HCAPLUS
DOCUMENT NUMBER: 131:227367
TITLE: **Immunization** with **amyloid** -.beta. attenuates **Alzheimer** **disease**-like pathology in the PDAPP mouse
AUTHOR(S): Schenk, Dale; Barbour, Robin; Dunn, Whitney; Gordon, Grace; Grajeda, Henry; Guido, Teresa; Hu, Kang; Huang, Jiping; Johnson-Wood, Kelly; Khan, Karen; Kholodenko, Dora; Lee, Mike; Liao, Zhenmei; Lieberburg, Ivan; Motter, Ruth; Mutter, Linda; Soriano, Ferdie; Shopp, George; Vasquez, Nicki; Vandevert, Christopher; Walker, Shannan; Wogulis, Mark; Yednock, Ted; Games, Dora; Seubert, Peter
CORPORATE SOURCE: Elan Pharmaceuticals, South San Francisco, CA, 94080, USA
SOURCE: Nature (London) (1999), 400(6740), 173-177
CODEN: NATUAS; ISSN: 0028-0836
PUBLISHER: Macmillan Magazines
DOCUMENT TYPE: Journal
LANGUAGE: English
AB **Amyloid**-.beta. peptide (A.beta.) seems to have a central role in the neuropathol. of **Alzheimer**'s **disease** (**AD**). Familial forms of the **disease** have been linked to mutations in the **amyloid** precursor protein (APP) and the presenilin genes. **Disease**-linked mutations in these genes result in increased prodn. of the 42-amino-acid form of the peptide (A.beta.42), which is the predominant form found in the **amyloid** plaques of **Alzheimer**'s **disease**.
The PDAPP transgenic mouse, which overexpresses mutant human APP (in which the amino acid at position 717 is phenylalanine instead of the normal valine), progressively develops many of the neuropathol. hallmarks of **Alzheimer**'s disease in an age- and

brain-region-dependent manner. In the present study, transgenic animals were **immunized** with A.beta.42, either before the onset of AD-type neuropathologies (at 6 wk of age) or at an older age (11 mo), when amyloid-.beta. deposition and several of the subsequent neuropathol. changes were well established. We report that **immunization** of the young animals essentially **prevented** the development of .beta.-amyloid-plaque formation, neuritic dystrophy and astrogliosis. **Treatment** of the older animals also markedly reduced the extent and progression of these AD-like neuropathologies. Our results raise the possibility that **immunization** with **amyloid** -.beta. may be effective in **preventing** and **treating Alzheimer's disease.**

IT **107761-42-2**, Glycopeptide (human clone 9-110 **amyloid** A4 peptide moiety)
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**immunization** with **amyloid**-.beta. attenuates **Alzheimer disease**-like pathol. in PDAPP mouse)

REFERENCE COUNT: 16 THERE ARE 16 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 26 OF 29 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1999:375416 HCAPLUS
DOCUMENT NUMBER: 131:27965
TITLE: **Prevention** and **treatment** of amyloidogenic **disease**, especially **Alzheimer's disease**, based on induction of anti-**amyloid** immune response
INVENTOR(S): Schenk, Dale B.
PATENT ASSIGNEE(S): Athena Neurosciences, Inc., USA
SOURCE: PCT Int. Appl., 113 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9927944	A1	19990610	WO 1998-US25386	19981130
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2312920	AA	19990610	CA 1998-2312920	19981130
AU 9917061	A1	19990616	AU 1999-17061	19981130
ZA 9810932	A	19990702	ZA 1998-10932	19981130
EP 1033996	A1	20000913	EP 1998-961833	19981130
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC,				

6-10

PT, IE, SI, LT, LV, FI, RO

BR 9815357	A	20001024	BR 1998-15357		19981130
JP 2002502802	T2	20020129	JP 2000-522929		19981130
NO 2000002784	A	20000731	NO 2000-2784		20000531
PRIORITY APPLN. INFO.:			US 1997-67740P	P	19971202
			US 1998-80970P	P	19980407
			WO 1998-US25386	W	19981130

AB The invention provides compns. and methods for **treatment** of amyloidogenic diseases. The methods entail administering an agent that induces a beneficial immune response against an amyloid deposit in the patient. The methods are particularly useful for prophylactic and **therapeutic treatment** of **Alzheimer**'s disease. In such methods, a suitable agent is A.beta. peptide or an antibody thereto.

IT **107761-42-2**, Glycopeptide (human clone 9-110 **amyloid** A4 peptide moiety)
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(**prevention** and **treatment** of amyloidogenic **disease**, esp. **Alzheimer**'s **disease**, based on induction of anti-**amyloid** immune response)

IT **131438-79-4 226917-46-0D**, IgG conjugates
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**prevention** and **treatment** of amyloidogenic **disease**, esp. **Alzheimer**'s **disease**, based on induction of anti-**amyloid** immune response)

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 27 OF 29 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1999:212578 HCAPLUS
DOCUMENT NUMBER: 131:57660
TITLE: Pro-inflammatory complement activation by the A.beta. peptide of **Alzheimer**'s disease is biologically significant and can be blocked by **vaccinia** virus complement **control** protein
AUTHOR(S): Daly, James; Kotwal, Girish J.
CORPORATE SOURCE: Department of Microbiology and Immunology, University of Louisville School of Medicine, Louisville, KY, 40292, USA
SOURCE: Neurobiology of Aging (1999), Volume Date 1998, 19(6), 619-627
CODEN: NEAGDO; ISSN: 0197-4580
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The **amyloid** plaque is the hallmark of **Alzheimer**'s **disease** (**AD**). The transmembrane domain and a portion of the C-terminus (A.beta.) of the amyloid precursor protein, are known to form the nucleus of the amyloid plaque. It has been demonstrated recently, using in vitro assays, that the A.beta. peptide can activate both the classical (antibody-

independent) and alternate pathways of complement activation. The proposed complement activation is due to the binding of A.beta. to the complement components Clq and C3, resp., which initiate formation of the proinflammatory C5a and C5b-9 membrane attack complex. In this report, the authors have investigated the in vitro findings for the likely complement-dependent proinflammatory properties of the **Alzheimer**'s disease A.beta. peptide. The authors have performed expts. using congenic C5-deficient and C5-sufficient mice injected with synthetic A.beta. and recombinant polypeptide (C-100) contg. A.beta.. Injection of C-100 into C5-sufficient mice induced a clear increase in the no. of polymorphonuclear cells (neutrophils) at the site of injection due to complement activation and the subsequent release of proinflammatory chemtoactic factors. In sharp contrast, the C5-deficient mice did not show any increase in cellular influx. The **vaccinia** virus complement **control** protein, an inhibitor of both the classical and alternate pathway can down-regulate the biol. significant activation of complement by A.beta., as demonstrated by an in vitro immunoassay. The **therapeutic** down-regulation of A.beta.-caused complement activation could greatly alleviate the progression of some of the chronic neurodegeneration characteristic of **Alzheimer**'s disease.

IT **134548-35-9**, 652-751-Glycoprotein (human clone .lambda.APCP168i4 **amyloid** A4 precursor protein moiety reduced)
RL: PRP (Properties)
(amino acid sequence; pro-inflammatory complement activation by A.beta. peptide of **Alzheimer**'s **disease** is biol. significant and can be blocked by **vaccinia** virus complement **control** protein)

REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 28 OF 29 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1991:598502 HCAPLUS
DOCUMENT NUMBER: 115:198502
TITLE: Assays and reagents for **amyloid** deposition and screening agents for **treating Alzheimer**'s **disease** amyloidosis
INVENTOR(S): Cordell, Barbara; Wolf, David
PATENT ASSIGNEE(S): California Biotechnology, Inc., USA
SOURCE: PCT Int. Appl., 38 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9104339	A1	19910404	WO 1990-US5155	19900912
W: AU, CA, JP				
RW: AT, BE, CH, DE, DK, ES, FR, GB, IT, LU, NL, SE				
CA 2065404	AA	19910319	CA 1990-2065404	19900912
AU 9064311	A1	19910418	AU 1990-64311	19900912

AU 641434	B2	19930923			
EP 493470	A1	19920708	EP 1990-914284	19900912	
R: AT, BE, CH, DE, DK, ES, FR, GB, IT, LI, LU, NL, SE					
JP 05502368	T2	19930428	JP 1990-513439	19900912	
US 5221607	A	19930622	US 1991-785142	19911029	
PRIORITY APPLN. INFO.:			US 1989-408767	19890918	
			WO 1990-US5155	19900912	

AB An in vitro tissue culture-based assay for **amyloid** deposition specific for **Alzheimer's disease** and immunol. assay reagents for screening agents capable of intervention in **Alzheimer's disease** amyloidosis are disclosed. Cell lines capable of expressing a gene encoding .beta.-amyloid protein under conditions suitable to produce the .beta.-amyloid protein as an insol., preamyloid aggregate are used. Amyloid plaque core DNA (pUV1:A42 and pUV1-A99) were constructed and used to make recombinant **vaccinia** viruses VV:A42 and VV:A99. Neuronal cell lines were infected with the recombinant viruses and then slides were prepd. for immunocytochem. The VV:A99 and VV:A42 infected cells displayed strong reactivity in the form of large deposit-like structures which were cell assocd.

IT **117313-01-6**, 597-695-Glycoprotein (human clone 9-110 **amyloid** A4 precursor protein moiety reduced)
RL: PRP (Properties)
(amino acid sequence of, **Alzheimer's disease** drugs screening in relation to)

L8 ANSWER 29 OF 29 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER:	1989:2189 HCAPLUS
DOCUMENT NUMBER:	110:2189
TITLE:	Diagnosis and **treatment** of **Alzheimer's disease**: cloning and expression of DNA encoding .beta.-**amyloid**-related protein
INVENTOR(S):	Greenberg, Barry D.; Fuller, Forrest H.; Ponte, Phyllis A.
PATENT ASSIGNEE(S):	California Biotechnology, Inc., USA
SOURCE:	PCT Int. Appl., 85 pp. CODEN: PIXXD2
DOCUMENT TYPE:	Patent
LANGUAGE:	English
FAMILY ACC. NUM. COUNT:	3
PATENT INFORMATION:	

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 8803951	A1	19880602	WO 1987-US2953	19871112
W: AU, JP, KR, US, US, US, US				
RW: AT, BE, CH, DE, FR, GB, IT, LU, NL, SE				
AU 8783290	A1	19880616	AU 1987-83290	19871112
AU 611122	B2	19910606		
EP 274826	A1	19880720	EP 1987-310029	19871112
EP 274826	B1	19980812		
R: ES, GR				
EP 332640	A1	19890920	EP 1987-907896	19871112
R: AT, BE, CH, DE, FR, GB, IT, LI, LU, NL, SE				
JP 02501796	T2	19900621	JP 1988-500173	19871112
JP 07079702	B4	19950830		
AT 169673	E	19980815	AT 1987-310029	19871112

ES 2120401	T3	19981101	ES 1987-310029	19871112
US 5220013	A	19930615	US 1989-444118	19891130

PRIORITY APPLN. INFO.:

US 1986-932193	19861117
US 1986-948376	19861231
US 1987-8810	19870130
US 1987-87002	19870818
WO 1987-US2953	19871112

AB DNA encoding human .beta.-amyloid-related protein is cloned and expressed in bacteria and mammalian cells. The DNA and antibodies to the protein may be used to diagnose **Alzheimer**'s disease. Immunogenic fragments of the protein may be used to **treat** the disease. A cDNA encoding amino acids 1-751 of .beta.-amyloid-related protein, and genomic DNA contg. DNA encoding the 1st 18 amino acids of the .beta.-amyloid core protein (according to Masters) preceded by methionine were cloned and sequenced. Plasmids for prodn. of .beta.-amyloid-related proteins in Escherichia coli and recombinant **vaccinia** virus for its prodn. in CV-1 cells were prepd. Antibodies were raised against the recombinant protein. The cloned DNA was used in Northern blotting expts. to distinguish genetic variants of .beta.-amyloid-related protein mRNA species.

IT **117312-63-7,** Glycopeptide (human clone .lambda.SMW9 amyloid A4 peptide moiety) **117312-93-3 117312-96-6** **117312-99-9,** 599-695-Glycoprotein (human clone 9-110 amyloid A4 precursor protein moiety reduced) **117313-00-5** **117313-01-6,** 597-695-Glycoprotein (human clone 9-110 amyloid A4 precursor protein moiety reduced) **117910-30-2,** Glycoprotein (human clone .lambda.APCP168i4 amyloid A4 precursor protein moiety reduced)
RL: PRP (Properties)
(amino acid sequence of and cloning in Escherichia coli of DNA encoding)

E1 THROUGH E47 ASSIGNED

FILE 'REGISTRY' ENTERED AT 12:20:52 ON 06 JAN 2003

L9 47 SEA FILE=REGISTRY ABB=ON PLU=ON (107761-42-2/BI OR 131438-79-4/BI OR 117313-01-6/BI OR 134500-80-4/BI OR 134548-35-9/BI OR 342877-52-5/BI OR 342877-55-8/BI OR 342877-58-1/BI OR 342877-61-6/BI OR 342877-63-8/BI OR 342877-66-1/BI OR 342877-69-4/BI OR 342877-71-8/BI OR 342877-73-0/BI OR 342877-74-1/BI OR 342877-75-2/BI OR 342877-97-8/BI OR 342878-00-6/BI OR 342878-03-9/BI OR 342878-06-2/BI OR 342878-09-5/BI OR 109770-29-8/BI OR 117312-63-7/BI OR 117312-93-3/BI OR 117312-96-6/BI OR 117312-99-9/BI OR 117313-00-5/BI OR 117910-30-2/BI OR 159647-22-0/BI OR 226707-64-8/BI OR 226917-46-0/BI OR 312263-74-4/BI OR 313474-75-8/BI OR 342896-25-7/BI OR 342896-48-4/BI OR 389151-08-0/BI OR 419018-03-4/BI OR 419018-23-8/BI OR 422613-26-1/BI OR 442915-40-4/BI OR 444137-68-2/BI OR 444137-69-3/BI OR 444137-70-6/BI OR 444309-74-4/BI OR 477826-92-9/BI OR 477826-93-0/BI OR 477826-96-3/BI)

=> s l9 and l1
L10 47 L9 AND L1

L10 ANSWER 1 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **477826-96-3** REGISTRY
CN L-Phenylalanine, L-.alpha.-aspartyl-L-alanyl-L-.alpha.-glutamyl-L-phenylalanyl-L-arginyl-L-histidyl-L-.alpha.-aspartyl-L-serylglycyl-L-tyrosyl-L-.alpha.-glutamyl-L-valyl-L-histidyl-L-histidyl-L-glutaminyl-L-lysyl-L-leucyl-L-valyl-L-phenylalanyl-L-phenylalanyl-L-alanyl-L-.alpha.-glutamyl-L-.alpha.-aspartyl-L-valylglycyl-L-seryl-L-asparaginyl-L-lysyl-L-lysyl-L-isoleucyl-L-seryl-L-isoleucyl-L-threonyl-L-.alpha.-glutamyl-L-isoleucyl-L-lysylglycyl-L-valyl-L-isoleucyl-L-valyl-L-histidyl-L-arginyl-L-isoleucyl-L-.alpha.-glutamyl-L-threonyl-L-isoleucyl-L-leucyl- (9CI) (CA INDEX NAME)
CI MAN
SQL 48

SEQ 1 DAEFRHDSGY EVHHQKLVFF AEDVGSNKKI SITEIKGVIV HRIETILF
====
HITS AT: 16-19

L10 ANSWER 2 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **477826-93-0** REGISTRY
CN L-Aspartic acid, L-.alpha.-aspartyl-L-alanyl-L-.alpha.-glutamyl-L-phenylalanyl-L-arginyl-L-histidyl-L-.alpha.-aspartyl-L-serylglycyl-L-tyrosyl-L-.alpha.-glutamyl-L-valyl-L-histidyl-L-histidyl-L-glutaminyl-L-lysyl-L-leucyl-L-valyl-L-phenylalanyl-L-phenylalanyl-L-alanyl-L-.alpha.-glutamyl-L-.alpha.-aspartyl-L-valylglycyl-L-seryl-L-asparaginyl-L-lysylglycylglycyl-L-phenylalanyl-L-phenylalanyl-L-leucyl-L-leucyl-L-threonyl-L-arginyl-L-isoleucyl-L-leucyl-L-threonyl-L-isoleucyl-L-prolyl-L-glutaminyl-L-seryl-L-leucyl- (9CI) (CA INDEX NAME)
CI MAN
SQL 45

SEQ 1 DAEFRHDSGY EVHHQKLVFF AEDVGSNKGG FFLLTRILTI PQSLD
====
HITS AT: 16-19

L10 ANSWER 3 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **477826-92-9** REGISTRY
CN INDEX NAME NOT YET ASSIGNED
CI MAN
SQL 42

SEQ 1 DAEFRHDSGY EVHHQKLVFF AEDVGSNKGA IIGLMVGGVV IA
====
HITS AT: 16-19

RELATED SEQUENCES AVAILABLE WITH SEQLINK

L10 ANSWER 4 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **444309-74-4** REGISTRY
CN L-Cysteine, L-.alpha.-aspartyl-L-alanyl-L-.alpha.-glutamyl-L-phenylalanyl-L-arginyl-L-histidyl-L-.alpha.-aspartyl-L-serylglycyl-L-tyrosyl-L-.alpha.-glutamyl-L-valyl-L-histidyl-L-histidyl-L-glutaminyl-L-lysyl-L-leucyl-L-valyl-L-phenylalanyl-L-phenylalanyl-L-alanyl-L-.alpha.-glutamyl-L-.alpha.-aspartyl-L-valylglycyl-L-seryl-L-asparaginylglycylglycyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 12: PN: WO02056907 PAGE: 227 claimed sequence

SQL 30

SEQ 1 DAEFRHDSGY EVHHQKLVFF AEDVGSNGGC
====

HITS AT: 16-19

REFERENCE 1: 137:139348

L10 ANSWER 5 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **444137-70-6** REGISTRY
CN L-Alanine, L-.alpha.-aspartyl-L-alanyl-L-.alpha.-glutamyl-L-phenylalanyl-L-arginyl-L-histidyl-L-.alpha.-aspartyl-L-serylglycyl-L-tyrosyl-L-.alpha.-glutamyl-L-valyl-L-histidyl-L-histidyl-L-glutaminyl-L-lysyl-L-leucyl-L-valyl-L-phenylalanyl-L-phenylalanyl-L-alanyl-L-.alpha.-glutamyl-L-.alpha.-aspartyl-L-valylglycyl-L-seryl-L-asparaginyl-L-lysylglycyl-L-alanyl-L-isoleucyl-L-isoleucylglycyl-L-leucyl-L-methionyl-L-valylglycylglycyl-L-valyl-L-valyl-L-isoleucyl-(9CI) (CA INDEX NAME)
OTHER NAMES:
CN 220: PN: WO02056907 SEQID: 220 unclaimed protein
CI MAN
SQL 42

SEQ 1 DAEFRHDSGY EVHHQKLVFF AEDVGSNKGA IIGLMVGGVV IA
====

HITS AT: 16-19

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 137:139348

L10 ANSWER 6 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **444137-69-3** REGISTRY
CN 219: PN: WO02056907 SEQID: 219 unclaimed protein (9CI) (CA INDEX NAME)
CI MAN
SQL 82

SEQ 1 GSGLTNIKTE EISEVKMDAE FRHDSGYEVH HQKLVFFAED VGSNKGAIIG
====
51 LMVGGVVIAT VIIITLVMLK KQYTSNHHGV VE
HITS AT: 33-36

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 137:139348

L10 ANSWER 7 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **444137-68-2** REGISTRY
CN 218: PN: WO02056907 SEQID: 218 unclaimed protein (9CI) (CA INDEX NAME)
CI MAN
SQL 770

SEQ 1 MLPGLALLLL AAWTARALEV PTDGNAGLLA EPQIAMFCGR LNMHMNVQNG
51 KWDSDPSGTK TCIDTKEGIL QYCQEVYPEL QITNVVEANQ PVTIQNWCKR
101 GRKQCKTHPH FVIPYRCLVG EFVSDALLVP DKCKFLHQER MDVCETHLHW
151 HTVAKETCSE KSTNLHDYGM LLPCGIDKFR GVEFVCCPLA EESDNVDSAD

```
       201 AEEDDSDVWW GGADTDYADG SEDKVVEVAE EEEVAEVEEE EADDDEDDED
       251 GDEVEEEAEE PYEEATERTT SIATTTTTTT ESVEEVVREV CSEQAETGPC
       301 RAMISRWYFD VTEGKCAPFF YGGCGGNRNN FDTEEYCMAV CGSAMSQSLL
       351 KTTQEPLARD PVKLPTTAAS TPDAVDKYLE TPGDENEHAH FQKAKERLEA
       401 KHRERMSQVM REWEEAERQA KNLPKADKKA VIQHFQEKVE SLEQEAANER
       451 QQLVETHMAR VEAMLNDRRR LALENYITAL QAVPPRPRHV FNMLKKYVRA
       501 EQKDRQHTLK HFEHVRMVDP KKAAQIRSQV MTHLRVIYER MNQSLSLLYN
       551 VPAVAEEIQD EVDELLQKEQ NYSDDVLANM ISEPRISYGN DALMPSLTET
       601 KTTVELLPVN GEFSLDDLQP WHSFGADSVP ANTENEVEPV DARPAADRGL
       651 TTRPGSGLTN IKTEEISEVK MDAEFRHDSG YEVHHQKLVF FAEDVGSNKG
                                                   ====
       701 AIIGLMVGGV VIATVIVITL VMLKKKQYTS IHHGVVEVDA AVTPEERHLS
       751 KMQQNGYENP TYKFFEQMQN
HITS AT:    687-690

**RELATED SEQUENCES AVAILABLE WITH SEQLINK**

REFERENCE   1:  137:139348

L10  ANSWER 8 OF 47  REGISTRY  COPYRIGHT 2003 ACS
RN   442915-40-4  REGISTRY
CN   D-Alanine, D-tyrosyl-D-.alpha.-glutamyl-D-valyl-D-histidyl-D-
     histidyl-D-glutaminyl-D-lysyl-D-leucyl-D-valyl-D-phenylalanyl-D-
     phenylalanyl- (9CI)  (CA INDEX NAME)
SQL  12

SEQ      1 YEVHHQKLVF FA
                ====
HITS AT:    7-10

**RELATED SEQUENCES AVAILABLE WITH SEQLINK**

REFERENCE   1:  137:108295

L10  ANSWER 9 OF 47  REGISTRY  COPYRIGHT 2003 ACS
RN   422613-26-1  REGISTRY
CN   L-Threonine, L-.alpha.-aspartyl-L-alanyl-L-.alpha.-glutamyl-L-
     phenylalanyl-L-arginyl-L-histidyl-L-.alpha.-aspartyl-L-serylglycyl-L-
     tyrosyl-L-.alpha.-glutamyl-L-valyl-L-histidyl-L-histidyl-L-
     glutaminyl-L-lysyl-L-leucyl-L-valyl-L-phenylalanyl-L-phenylalanyl-L-
     alanyl-L-.alpha.-glutamyl-L-.alpha.-aspartyl-L-valylglycyl-L-seryl-L-
     asparaginyl-L-lysylglycyl-L-alanyl-L-isoleucyl-L-isoleucylglycyl-L-
     leucyl-L-methionyl-L-valylglycylglycyl-L-valyl-L-valyl-L-isoleucyl-L-
     alanyl- (9CI)  (CA INDEX NAME)
OTHER NAMES:
CN   2: PN: WO0236614 SEQID: 2 claimed protein
CI   MAN
SQL  43

SEQ      1 DAEFRHDSGY EVHHQKLVFF AEDVGSNKGA IIGLMVGGVV IAT
                       ====
HITS AT:    16-19

**RELATED SEQUENCES AVAILABLE WITH SEQLINK**

REFERENCE   1:  136:363863

L10  ANSWER 10 OF 47  REGISTRY  COPYRIGHT 2003 ACS
```

RN **419018-23-8** REGISTRY
CN L-Threonine, L-.alpha.-aspartyl-L-alanyl-L-.alpha.-glutamyl-L-phenylalanyl-L-arginyl-L-histidyl-L-.alpha.-aspartyl-L-serylglycyl-L-tyrosyl-L-.alpha.-glutamyl-L-valyl-L-histidyl-L-histidyl-L-glutaminyl-L-lysyl-L-leucyl-L-valyl-L-phenylalanyl-L-phenylalanyl-L-alanyl-L-.alpha.-glutamyl-L-.alpha.-aspartyl-L-valylglycyl-L-seryl-L-asparaginyl-L-lysylglycyl-L-alanyl-L-isoleucyl-L-isoleucylglycyl-L-leucyl-L-methionyl-L-valylglycylglycyl-L-valyl-L-valyl-L-isoleucyl-L-alanyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 2: PN: US20020052311 SEQID: 3 unclaimed protein
CI MAN
SQL 43

SEQ 1 DAEFRHDSGY EVHHQKLVFF AEDVGSNKGA IIGLMVGGVV IAT
====
HITS AT: 16-19

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 136:345817

L10 ANSWER 11 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **419018-03-4** REGISTRY
CN Glycopeptide (human clone 9-110 amyloid A4 peptide moiety) fusion protein with heat-shock protein HSP 70 (synthetic) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 2: PN: WO0234777 SEQID: 2 claimed protein
CI MAN
SQL 670

SEQ 1 MDAEFRHDSG YEVHHQKLVF FAEDVGSNKG AIIGLMVGGV VIAGSMARAV
====
51 GIDLGTTNSV VSVLEGGDPV VVANSEGSRT TPSIVAFARN GEVLVGQPAK
101 NQAVTNVDRT VRSVKRHMGS DWSIEIDGKK YTAPEISARI LMKLKRDAEA
151 YLGEDITDAV ITTPAYFNDA QRQATKDAGQ IAGLNVLRIV NEPTAAALAY
201 GLDKGEKEQR ILVFDLGGGT FDVSLLEIGE GVVEVRATSG DNHLGGDDWD
251 QRVVDWLVDK FKGTSGIDLT KDKMAMQRLR EAAEKAKIEL SSSQSTSINL
301 PYITVDADKN PLFLDEQLTR AEFQRITQDL LDRTRKPFQS VIADTGISVS
351 EIDHVVLVGG STRMPAVTDL VKELTGGKEP NKGVNPDEVV AVGAALQAGV
401 LKGEVKDVLL LDVTPLSLGI ETKGGVMTRL IERNTTIPTK RSETFTTADD
451 NQPSVQIQVY QGEREIAAHN KLLGSFELTG IPPAPRGIPQ IEVTFDIDAN
501 GIVHVTAKDK GTGKENTIRI QEGSGLSKED IDRMIKDAEA HAEEDRKRRE
551 EADVRNQAET LVYQTEKFVK EQREAEGGSK VPEDTLNKVD AAVAEAKAAL
601 GGSDISAIKS AMEKLGQESQ ALGQAIYEAA QAASQATGAA HPGGEPGGAH
651 PGSADDVVDA EVVDDGREAK
HITS AT: 17-20

REFERENCE 1: 136:339487

L10 ANSWER 12 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **389151-08-0** REGISTRY
CN L-Alanine, L-.alpha.-aspartyl-L-alanyl-L-.alpha.-glutamyl-L-phenylalanyl-L-arginyl-L-histidyl-L-.alpha.-aspartyl-L-serylglycyl-L-tyrosyl-L-.alpha.-glutamyl-L-valyl-L-histidyl-L-histidyl-L-glutaminyl-L-lysyl-L-leucyl-L-valyl-L-phenylalanyl-L-phenylalanyl-L-alanylglycyl-L-.alpha.-aspartyl-L-valylglycyl-L-seryl-L-asparaginyl-

L-lysylglycyl-L-alanyl-L-isoleucyl-L-isoleucylglycyl-L-leucyl-L-methionyl-L-valylglycylglycyl-L-valyl-L-valyl-L-isoleucyl- (9CI) (CA INDEX NAME)
CI MAN
SQL 42

SEQ 1 DAEFRHDSGY EVHHQKLVFF AGDVGSNKGA IIGLMVGGVV IA
====

HITS AT: 16-19

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 136:112669

L10 ANSWER 13 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **342896-48-4** REGISTRY
CN D-Methionine, D-.alpha.-aspartyl-D-alanyl-D-.alpha.-glutamyl-D-phenylalanyl-D-arginyl-D-histidyl-D-.alpha.-aspartyl-D-serylglycyl-D-tyrosyl-D-.alpha.-glutamyl-D-valyl-D-histidyl-D-histidyl-D-glutaminyl-D-lysyl-D-leucyl-D-valyl-D-phenylalanyl-D-phenylalanyl-D-alanyl-D-.alpha.-glutamyl-D-.alpha.-aspartyl-D-valylglycyl-D-seryl-D-asparaginyl-D-lysylglycyl-D-alanyl-D-isoleucyl-D-isoleucylglycyl-D-leucyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 3: PN: WO0139796 SEQID: 3 claimed sequence
CI MAN
SQL 35

SEQ 1 DAEFRHDSGY EVHHQKLVFF AEDVGSNKGA IIGLM
====

HITS AT: 16-19

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 135:18553

L10 ANSWER 14 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **342896-25-7** REGISTRY
CN D-Alanine, D-.alpha.-aspartyl-D-alanyl-D-.alpha.-glutamyl-D-phenylalanyl-D-arginyl-D-histidyl-D-.alpha.-aspartyl-D-serylglycyl-D-tyrosyl-D-.alpha.-glutamyl-D-valyl-D-histidyl-D-histidyl-D-glutaminyl-D-lysyl-D-leucyl-D-valyl-D-phenylalanyl-D-phenylalanyl-D-alanyl-D-.alpha.-glutamyl-D-.alpha.-aspartyl-D-valylglycyl-D-seryl-D-asparaginyl-D-lysylglycyl-D-alanyl-D-isoleucyl-D-isoleucylglycyl-D-leucyl-D-methionyl-D-valylglycylglycyl-D-valyl-D-valyl-D-isoleucyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 1: PN: WO0139796 SEQID: 1 claimed sequence
CI MAN
SQL 42

SEQ 1 DAEFRHDSGY EVHHQKLVFF AEDVGSNKGA IIGLMVGGVV IA
====

HITS AT: 16-19

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 135:18553

L10 ANSWER 15 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **342878-09-5** REGISTRY
CN D-Alaninamide, D-lysyl-D-leucyl-D-valyl-D-phenylalanyl-(2R)-2-phenylglycyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 62: PN: WO0139796 SEQID: 62 claimed protein
SQL 6

SEQ 1 KLVFXA
====
HITS AT: 1-4

REFERENCE 1: 137:108295

REFERENCE 2: 135:18553

L10 ANSWER 16 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **342878-06-2** REGISTRY
CN D-Alaninamide, D-lysyl-D-leucyl-D-valyl-D-phenylalanyl-3-cyclohexyl-D-alanyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 59: PN: WO0139796 SEQID: 59 claimed protein
SQL 6

SEQ 1 KLVFAA
====
HITS AT: 1-4

REFERENCE 1: 137:108295

REFERENCE 2: 135:18553

L10 ANSWER 17 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **342878-03-9** REGISTRY
CN D-Alaninamide, D-lysyl-D-leucyl-D-valyl-D-phenylalanyl-3-(2-thienyl)-D-alanyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 56: PN: WO0139796 SEQID: 56 claimed protein
SQL 6

SEQ 1 KLVFXA
====
HITS AT: 1-4

REFERENCE 1: 137:108295

REFERENCE 2: 135:18553

L10 ANSWER 18 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **342878-00-6** REGISTRY
CN D-Alaninamide, D-lysyl-D-leucyl-D-valyl-D-phenylalanyl-D-tyrosyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 53: PN: WO0139796 SEQID: 53 claimed protein
SQL 6

SEQ 1 KLVFYA

```
          ====
HITS AT:   1-4

REFERENCE   1:  137:108295

REFERENCE   2:  135:18553

L10  ANSWER 19 OF 47  REGISTRY  COPYRIGHT 2003 ACS
RN   342877-97-8  REGISTRY
CN   D-Alaninamide, D-lysyl-D-leucyl-D-valyl-D-phenylalanyl-D-tryptophyl-
     (9CI)  (CA INDEX NAME)
OTHER NAMES:
CN   50: PN: WO0139796 SEQID: 50 claimed protein
SQL  6

SEQ      1 KLVFWA
           ====
HITS AT:   1-4

REFERENCE   1:  137:108295

REFERENCE   2:  135:18553

L10  ANSWER 20 OF 47  REGISTRY  COPYRIGHT 2003 ACS
RN   342877-75-2  REGISTRY
CN   D-Glutamine, D-histidyl-D-histidyl-D-glutaminyl-D-lysyl-D-leucyl-D-
     valyl-D-phenylalanyl-D-phenylalanyl-D-alanyl- (9CI)  (CA INDEX NAME)
OTHER NAMES:
CN   27: PN: WO0139796 SEQID: 27 claimed protein
SQL  10

SEQ      1 HHQKLVFFAQ
              ====
HITS AT:   4-7

**RELATED SEQUENCES AVAILABLE WITH SEQLINK**

REFERENCE   1:  137:108295

REFERENCE   2:  135:18553

L10  ANSWER 21 OF 47  REGISTRY  COPYRIGHT 2003 ACS
RN   342877-74-1  REGISTRY
CN   D-Glutamamide, D-lysyl-D-leucyl-D-valyl-D-phenylalanyl-D-
     phenylalanyl-D-alanyl- (9CI)  (CA INDEX NAME)
OTHER NAMES:
CN   26: PN: WO0139796 SEQID: 26 claimed protein
SQL  7

SEQ      1 KLVFFAQ
           ====
HITS AT:   1-4

**RELATED SEQUENCES AVAILABLE WITH SEQLINK**

REFERENCE   1:  137:108295

REFERENCE   2:  135:18553
```

L10 ANSWER 22 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **342877-73-0** REGISTRY
CN D-Glutamine, D-lysyl-D-leucyl-D-valyl-D-phenylalanyl-D-phenylalanyl-D-alanyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 25: PN: WO0139796 SEQID: 25 claimed protein
SQL 7

SEQ 1 KLVFFAQ
====
HITS AT: 1-4

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 137:108295

REFERENCE 2: 135:18553

L10 ANSWER 23 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **342877-71-8** REGISTRY
CN D-Phenylalaninamide, D-lysyl-D-leucyl-D-valyl-D-phenylalanyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 23: PN: WO0139796 SEQID: 23 claimed protein
SQL 5

SEQ 1 KLVFF
====
HITS AT: 1-4

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 137:108295

REFERENCE 2: 135:18553

L10 ANSWER 24 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **342877-69-4** REGISTRY
CN D-Phenylalaninamide, D-lysyl-D-leucyl-D-valyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 21: PN: WO0139796 SEQID: 21 claimed protein
SQL 4

SEQ 1 KLVF
====
HITS AT: 1-4

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 137:108295

REFERENCE 2: 135:18553

L10 ANSWER 25 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **342877-66-1** REGISTRY
CN D-Alaninamide, D-lysyl-D-leucyl-D-valyl-D-phenylalanyl-D-

phenylalanyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 18: PN: WO0139796 SEQID: 18 claimed protein
SQL 6

SEQ 1 KLVFFA
====
HITS AT: 1-4

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 137:108295

REFERENCE 2: 135:18553

L10 ANSWER 26 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **342877-63-8** REGISTRY
CN D-Phenylalanine, D-lysyl-D-leucyl-D-valyl-D-phenylalanyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 15: PN: WO0139796 SEQID: 15 claimed protein
SQL 5

SEQ 1 KLVFF
====
HITS AT: 1-4

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 137:167565

REFERENCE 2: 137:108295

REFERENCE 3: 135:18553

L10 ANSWER 27 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **342877-61-6** REGISTRY
CN D-Phenylalanine, D-lysyl-D-leucyl-D-valyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 13: PN: WO0139796 SEQID: 13 claimed protein
SQL 4

SEQ 1 KLVF
====
HITS AT: 1-4

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 137:108295

REFERENCE 2: 135:18553

L10 ANSWER 28 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **342877-58-1** REGISTRY
CN D-Alanine, D-lysyl-D-lysyl-D-leucyl-D-valyl-D-phenylalanyl-D-phenylalanyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 10: PN: WO0139796 SEQID: 10 claimed protein

SQL 7

SEQ 1 KKLVFFA
====
HITS AT: 2-5

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 137:108295

REFERENCE 2: 135:18553

L10 ANSWER 29 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **342877-55-8** REGISTRY
CN D-Alanine, D-lysyl-D-leucyl-D-valyl-D-phenylalanyl-D-phenylalanyl-
(9CI) (CA INDEX NAME)
OTHER NAMES:
CN 7: PN: WO0139796 SEQID: 7 claimed protein
SQL 6

SEQ 1 KLVFFA
====
HITS AT: 1-4

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 137:108295

REFERENCE 2: 136:139864

REFERENCE 3: 135:18553

L10 ANSWER 30 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **342877-52-5** REGISTRY
CN D-Lysine, D-.alpha.-aspartyl-D-alanyl-D-.alpha.-glutamyl-D-
phenylalanyl-D-arginyl-D-histidyl-D-.alpha.-aspartyl-D-serylglycyl-D-
tyrosyl-D-.alpha.-glutamyl-D-valyl-D-histidyl-D-histidyl-D-
glutaminyl-D-lysyl-D-leucyl-D-valyl-D-phenylalanyl-D-phenylalanyl-D-
alanyl-D-.alpha.-glutamyl-D-.alpha.-aspartyl-D-valylglycyl-D-seryl-D-
asparaginyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 4: PN: WO0139796 SEQID: 4 claimed protein
SQL 28

SEQ 1 DAEFRHDSGY EVHHQKLVFF AEDVGSNK
====
HITS AT: 16-19

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 137:108295

REFERENCE 2: 135:18553

L10 ANSWER 31 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **313474-75-8** REGISTRY
CN Glycinamide, L-cysteinyl-L-tyrosyl-L-.alpha.-glutamyl-L-valyl-L-
histidyl-L-histidyl-L-glutaminyl-L-lysyl-L-leucyl-L-valyl-L-

phenylalanyl-L-phenylalanyl-L-alanyl-L-.alpha.-glutamyl-L-.alpha.-aspartyl-L-valyl- (9CI) (CA INDEX NAME)
SQL 17

SEQ 1 CYEVHHQKLV FFAEDVG
=== =
HITS AT: 8-11

REFERENCE 1: 134:55503

L10 ANSWER 32 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **312263-74-4** REGISTRY
CN L-Histidine, L-asparaginyl-L-histidyl-L-histidyl-L-histidyl-L-glutaminyl-L-lysyl-L-leucyl-L-valyl-L-phenylalanyl-L-phenylalanyl-L-alanyl-L-.alpha.-glutamyl-L-.alpha.-aspartyl-L-valylglycyl-L-seryl-L-asparaginyl-L-lysylglycylglycyl-L-cysteinyl-L-cysteinyl-L-glutaminyl-L-glutaminyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 33: PN: WO0072876 PAGE: 74 unclaimed sequence
SQL 25

SEQ 1 NHHHQKLVFF AEDVGSNKGG CCQQH
====
HITS AT: 6-9

REFERENCE 1: 134:28431

L10 ANSWER 33 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **226917-46-0** REGISTRY
CN L-Cysteine, N-acetyl-L-histidyl-L-histidyl-L-glutaminyl-L-lysyl-L-leucyl-L-valyl-L-phenylalanyl-L-phenylalanyl-L-alanyl-L-.alpha.-glutamyl-L-.alpha.-aspartyl-L-valylglycyl-L-seryl-L-asparaginyl-L-lysylglycylglycyl- (9CI) (CA INDEX NAME)
SQL 19

SEQ 1 HHQKLVFFAE DVGSNKGGC
====
HITS AT: 4-7

REFERENCE 1: 131:27965

L10 ANSWER 34 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **226707-64-8** REGISTRY
CN D-Valine, D-.alpha.-aspartyl-D-alanyl-D-.alpha.-glutamyl-D-phenylalanyl-D-arginyl-D-histidyl-D-.alpha.-aspartyl-D-serylglycyl-D-tyrosyl-D-.alpha.-glutamyl-D-valyl-D-histidyl-D-histidyl-D-glutaminyl-D-lysyl-D-leucyl-D-valyl-D-phenylalanyl-D-phenylalanyl-D-alanyl-D-.alpha.-glutamyl-D-.alpha.-aspartyl-D-valylglycyl-D-seryl-D-asparaginyl-D-lysylglycyl-D-alanyl-D-isoleucyl-D-isoleucylglycyl-D-leucyl-D-methionyl-D-valylglycylglycyl-D-valyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 2: PN: WO0139796 SEQID: 2 claimed protein
CI MAN
SQL 40

SEQ 1 DAEFRHDSGY EVHHQKLVFF AEDVGSNKGA IIGLMVGGVV
====

HITS AT: 16-19

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 135:18553

REFERENCE 2: 131:30581

L10 ANSWER 35 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **159647-22-0** REGISTRY
CN L-Valine, L-lysyl-L-leucyl-L-valyl-L-phenylalanyl-L-phenylalanyl-L-alanyl-L-.alpha.-glutamyl-L-.alpha.-aspartyl- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN L-Valine, N-[N-[N-[N-[N-[N-(N-L-lysyl-L-leucyl)-L-valyl]-L-phenylalanyl]-L-phenylalanyl]-L-alanyl]-L-.alpha.-glutamyl]-L-.alpha.-aspartyl]-
SQL 9

SEQ 1 KLVFFAEDV
====
HITS AT: 1-4

REFERENCE 1: 136:112669

REFERENCE 2: 122:78292

L10 ANSWER 36 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **134548-35-9** REGISTRY
CN 652-751-Amyloid precursor protein (human clone .lambda.APCP168i4) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 1: PN: WO0183811 SEQID: 1 claimed protein
CN 4: PN: WO0043027 SEQID: 14 unclaimed protein
CN 596-695-Glycoprotein (human clone 9-110 amyloid A4 precursor)
CN 652-751-Glycoprotein (human clone .lambda.APCP168i4 amyloid A4 precursor protein moiety reduced)
CN Amyloid precursor protein (human clone pAPPc C-terminal fragment)
CI MAN
SQL 100

SEQ 1 MDAEFRHDSG YEVHHQKLVF FAEDVGSNKG AIIGLMVGGV VIATVIVITL
====
51 VMLKKKQYTS IHHGVVEVDA AVTPEERHLS KMQQNGYENP TYKFFEQMQN
HITS AT: 17-20

REFERENCE 1: 135:354682

REFERENCE 2: 133:129886

REFERENCE 3: 131:57660

REFERENCE 4: 128:307169

REFERENCE 5: 115:23683

L10 ANSWER 37 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **134500-80-4** REGISTRY
CN L-Threonine, L-.alpha.-aspartyl-L-alanyl-L-.alpha.-glutamyl-L-

phenylalanyl-L-arginyl-L-histidyl-L-.alpha.-aspartyl-L-serylglycyl-L-tyrosyl-L-.alpha.-glutamyl-L-valyl-L-histidyl-L-histidyl-L-glutaminyl-L-lysyl-L-leucyl-L-valyl-L-phenylalanyl-L-phenylalanyl-L-alanyl-L-.alpha.-glutamyl-L-.alpha.-aspartyl-L-valylglycyl-L-seryl-L-asparaginyl-L-lysylglycyl-L-alanyl-L-isoleucyl-L-isoleucylglycyl-L-leucyl-L-methionyl-L-valylglycylglycyl-L-valyl-L-valyl-L-isoleucyl-L-alanyl- (9CI) (CA INDEX NAME)

OTHER NAMES:

CN .beta.-Amyloid A4(1-43)

CN 1: PN: US5985242 SEQID: 1 unclaimed protein

CN 1: PN: US6319498 SEQID: 1 unclaimed protein

CN 1: PN: WO0028331 PAGE: 32 unclaimed protein

CN 1: PN: WO0118169 SEQID: 3 claimed protein

CN 274: PN: WO0069900 SEQID: 954 unclaimed protein

CN 2: PN: WO0042166 PAGE: 29 unclaimed protein

CN 3: PN: US5985242 SEQID: 1 claimed protein

CN 3: PN: WO0142306 SEQID: 4 unclaimed protein

CN 596-638-Glycoprotein (human clone 9-110 amyloid A4 precursor protein moiety reduced)

CI MAN

SQL 43

SEQ 1 DAEFRHDSGY EVHHQKLVFF AEDVGSNKGA IIGLMVGGVV IAT

====

HITS AT: 16-19

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 136:245164

REFERENCE 2: 136:181848

REFERENCE 3: 136:148989

REFERENCE 4: 136:116689

REFERENCE 5: 136:665

REFERENCE 6: 135:255546

REFERENCE 7: 135:120638

REFERENCE 8: 135:60157

REFERENCE 9: 134:305336

REFERENCE 10: 134:236224

L10 ANSWER 38 OF 47 REGISTRY COPYRIGHT 2003 ACS

RN **131438-79-4** REGISTRY

CN L-Valine, L-.alpha.-aspartyl-L-alanyl-L-.alpha.-glutamyl-L-phenylalanyl-L-arginyl-L-histidyl-L-.alpha.-aspartyl-L-serylglycyl-L-tyrosyl-L-.alpha.-glutamyl-L-valyl-L-histidyl-L-histidyl-L-glutaminyl-L-lysyl-L-leucyl-L-valyl-L-phenylalanyl-L-phenylalanyl-L-alanyl-L-.alpha.-glutamyl-L-.alpha.-aspartyl-L-valylglycyl-L-seryl-L-asparaginyl-L-lysylglycyl-L-alanyl-L-isoleucyl-L-isoleucylglycyl-L-leucyl-L-methionyl-L-valylglycylglycyl-L-valyl- (9CI) (CA INDEX NAME)

OTHER NAMES:
CN .beta.-Amyloid peptide(1-40)
CN .beta.-Amyloid protein(1-40)
CN 1: PN: JP2001247600 SEQID: 1 unclaimed protein
CN 1: PN: WO0152890 SEQID: 1 claimed protein
CN 276: PN: WO0069900 SEQID: 956 unclaimed protein
CN 2: PN: WO0142306 SEQID: 2 unclaimed protein
CN 54: PN: WO0038706 SEQID: 14 unclaimed protein
CN 7: PN: US6043283 FIGURE: 17 claimed protein
CN Amyloid .beta. peptide(1-40) (synthetic)
CN Human .beta.-amyloid peptide-(1-40)
CI MAN
SQL 40

SEQ 1 DAEFRHDSGY EVHHQKLVFF AEDVGSNKGA IIGLMVGGVV
====

HITS AT: 16-19

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 138:11335

REFERENCE 2: 138:3197

REFERENCE 3: 138:3183

REFERENCE 4: 137:367804

REFERENCE 5: 137:350697

REFERENCE 6: 137:350612

REFERENCE 7: 137:336165

REFERENCE 8: 137:336164

REFERENCE 9: 137:325632

REFERENCE 10: 137:308734

L10 ANSWER 39 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **117910-30-2** REGISTRY
CN Glycoprotein (human clone .lambda.APCP168i4 amyloid A4 precursor protein moiety reduced) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 2: PN: WO0182967 SEQID: 4 unclaimed protein
CN 57: PN: WO0149098 SEQID: 57 claimed sequence
CN 57: PN: WO0150829 SEQID: 57 claimed sequence
CN 63: PN: WO0149097 SEQID: 57 claimed protein
CN 65: PN: WO0123533 SEQID: 57 unclaimed protein
CN Amyloid precursor protein (human 751-amino acid isoform)
CN Amyloid precursor protein (human isoform APP751)
CI MAN
SQL 751

SEQ 1 MLPGLALLLL AAWTARALEV PTDGNAGLLA EPQIAMFCGR LNMHMNVQNG
51 KWDSDPSGTK TCIDTKEGIL QYCQEVYPEL QITNVVEANQ PVTIQNWCKR
101 GRKQCKTHPH FVIPYRCLVG EFVSDALLVP DKCKFLHQER MDVCETHLHW

```
       151 HTVAKETCSE KSTNLHDYGM LLPCGIDKFR GVEFVCCPLA EESDNVDSAD
       201 AEEDDSDVWW GGADTDYADG SEDKVVEVAE EEEVAEVEEE EADDDEDDED
       251 GDEVEEEAEE PYEEATERTT SIATTTTTTT ESVEEVVREV CSEQAETGPC
       301 RAMISRWYFD VTEGKCAPFF YGGCGGNRNN FDTEEYCMAV CGSAIPTTAA
       351 STPDAVDKYL ETPGDENEHA HFQKAKERLE AKHRERMSQV MREWEEAERQ
       401 AKNLPKADKK AVIQHFQEKV ESLEQEAANE RQQLVETHMA RVEAMLNDRR
       451 RLALENYITA LQAVPPRPRH VFNMLKKYVR AEQKDRQHTL KHFEHVRMVD
       501 PKKAAQIRSQ VMTHLRVIYE RMNQSLSLLY NVPAVAEEIQ DEVDELLQKE
       551 QNYSDDVLAN MISEPRISYG NDALMPSLTE TKTTVELLPV NGEFSLDDLQ
       601 PWHSFGADSV PANTENEVEP VDARPAADRG LTTRPGSGLT NIKTEEISEV
       651 KMDAEFRHDS GYEVHHQKLV FFAEDVGSNK GAIIGLMVGG VVIATVIVIT
                           === =
       701 LVMLKKKQYT SIHHGVVEVD AAVTPEERHL SKMQQNGYEN PTYKFFEQMQ
       751 N
HITS AT:   668-671

**RELATED SEQUENCES AVAILABLE WITH SEQLINK**

REFERENCE   1:  135:352818

REFERENCE   2:  135:104271

REFERENCE   3:  135:104269

REFERENCE   4:  135:104268

REFERENCE   5:  134:277406

REFERENCE   6:  126:127855

REFERENCE   7:  118:17443

REFERENCE   8:  116:253564

REFERENCE   9:  115:189727

REFERENCE  10:  115:23683

L10  ANSWER 40 OF 47  REGISTRY  COPYRIGHT 2003 ACS
RN   117313-01-6  REGISTRY
CN   597-695-Glycoprotein (human clone 9-110 amyloid A4 precursor protein
     moiety reduced) (9CI)  (CA INDEX NAME)
OTHER NAMES:
CN   Glycoprotein, amyloid A4, pre-(human 99-amino acid carboxyl terminal
     fragment)
CI   MAN
SQL  99

SEQ      1 DAEFRHDSGY EVHHQKLVFF AEDVGSNKGA IIGLMVGGVV IATVIVITLV
                     ====
        51 MLKKKQYTSI HHGVVEVDAA VTPEERHLSK MQQNGYENPT YKFFEQMQN
HITS AT:   16-19

REFERENCE   1:  123:282774

REFERENCE   2:  122:209218

REFERENCE   3:  116:253564
```

REFERENCE 4: 115:198502

REFERENCE 5: 110:2189

L10 ANSWER 41 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **117313-00-5** REGISTRY
CN 599-695-Glycoprotein (human clone 9-110 amyloid A4 precursor protein moiety reduced), 599-L-methionine- (9CI) (CA INDEX NAME)
CI MAN
SQL 97

SEQ 1 MFRHDSGYEV HHQKLVFFAE DVGSNKGAII GLMVGGVVIA TVIVITLVML
====
51 KKKQYTSIHH GVVEVDAAVT PEERHLSKMQ QNGYENPTYK FFEQMQN
HITS AT: 14-17

REFERENCE 1: 110:2189

L10 ANSWER 42 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **117312-99-9** REGISTRY
CN 599-695-Glycoprotein (human clone 9-110 amyloid A4 precursor protein moiety reduced) (9CI) (CA INDEX NAME)
CI MAN
SQL 97

SEQ 1 EFRHDSGYEV HHQKLVFFAE DVGSNKGAII GLMVGGVVIA TVIVITLVML
====
51 KKKQYTSIHH GVVEVDAAVT PEERHLSKMQ QNGYENPTYK FFEQMQN
HITS AT: 14-17

REFERENCE 1: 110:2189

L10 ANSWER 43 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **117312-96-6** REGISTRY
CN 603-695-Glycoprotein (human clone 9-110 amyloid A4 precursor protein moiety reduced), 603-L-methionine- (9CI) (CA INDEX NAME)
CI MAN
SQL 93

SEQ 1 MSGYEVHHQK LVFFAEDVGS NKGAIIGLMV GGVVIATVIV ITLVMLKKKQ
= ===
51 YTSIHHGVVE VDAAVTPEER HLSKMQQNGY ENPTYKFFEQ MQN
HITS AT: 10-13

REFERENCE 1: 110:2189

L10 ANSWER 44 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **117312-93-3** REGISTRY
CN 604-695-Glycoprotein (human clone 9-110 amyloid A4 precursor protein moiety reduced), 604-L-methionine- (9CI) (CA INDEX NAME)
CI MAN
SQL 92

SEQ 1 MGYEVHHQKL VFFAEDVGSN KGAIIGLMVG GVVIATVIVI TLVMLKKKQY
== ==
51 TSIHHGVVEV DAAVTPEERH LSKMQQNGYE NPTYKFFEQM QN
HITS AT: 9-12

REFERENCE 1: 110:2189

L10 ANSWER 45 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **117312-63-7** REGISTRY
CN Glycopeptide (human clone .lambda.SMW9 amyloid A4 peptide moiety) (9CI) (CA INDEX NAME)
CI MAN
SQL 42

SEQ 1 EFGHDSGFEV RHQKLVFFAE DVGSNKGAII GLMVGGVVIA TV
====
HITS AT: 14-17

REFERENCE 1: 110:2189

L10 ANSWER 46 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **109770-29-8** REGISTRY
CN 1-28-Glycopeptide (human clone 9-110 amyloid A4 peptide moiety) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 1-28-Peptide .beta. (human amyloid)
CN 12: PN: WO0228351 SEQID: 12 unclaimed sequence
CN 279: PN: WO0069900 SEQID: 959 unclaimed sequence
CN 285: PN: WO0069900 SEQID: 965 unclaimed sequence
CN 2: PN: WO0069456 SEQID: 11 unclaimed sequence
CN 2: PN: WO0197841 SEQID: 2 unclaimed sequence
CN 312: PN: WO0069900 SEQID: 992 unclaimed sequence
CN 37: PN: WO0200883 PAGE: 25 unclaimed sequence
CN 3: PN: WO0200885 SEQID: 3 unclaimed sequence
CN 3: PN: WO0218585 SEQID: 2 unclaimed sequence
CN 6: PN: US6043283 FIGURE: 17 claimed protein
CN 7: PN: WO02053761 SEQID: 7 unclaimed sequence
CN 8: PN: WO0238177 SEQID: 7 claimed sequence
CN 98: PN: WO0109309 PAGE: 23 unclaimed sequence
CN Human .beta.-amyloid peptide(1-28)
SQL 28

SEQ 1 DAEFRHDSGY EVHHQKLVFF AEDVGSNK
====
HITS AT: 16-19

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 137:306930

REFERENCE 2: 137:107986

REFERENCE 3: 137:103906

REFERENCE 4: 137:75823

REFERENCE 5: 136:384964

REFERENCE 6: 136:320642

REFERENCE 7: 136:290805

REFERENCE 8: 136:227876

REFERENCE 9: 136:98025

REFERENCE 10: 136:80862

L10 ANSWER 47 OF 47 REGISTRY COPYRIGHT 2003 ACS
RN **107761-42-2** REGISTRY
CN Glycopeptide (human clone 9-110 amyloid A4 peptide moiety) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 181: PN: WO0185208 SEQID: 174 unclaimed protein
CN 1: PN: WO0068263 FIGURE: 1 claimed protein
CN 1: PN: WO0069456 SEQID: 10 unclaimed protein
CN 1: PN: WO0182967 SEQID: 2 unclaimed protein
CN 1: PN: WO0246222 SEQID: 1 claimed protein
CN 21: PN: WO0142306 SEQID: 3 unclaimed protein
CN 275: PN: WO0069900 SEQID: 955 unclaimed protein
CN 2: PN: JP2001247600 SEQID: 2 unclaimed protein
CN 2: PN: WO0152890 SEQID: 2 claimed protein
CN 2: PN: WO0246222 SEQID: 1 claimed protein
CN 34: PN: WO0072876 PAGE: 24 unclaimed protein
CN 52: PN: WO0038706 SEQID: 10 unclaimed protein
CN 5: PN: WO0132694 SEQID: 5 claimed protein
CN 6: PN: US6043283 FIGURE: 17 claimed protein
CN 97: PN: WO0109309 PAGE: 23 unclaimed protein
CN Amyloid .beta. 1-42
CN Glycoprotein, amyloid A4 (human clone .lambda.APCP168i4 N-terminal 42-amino-acid fragment)
CN Human .beta.-amyloid peptide-(1-42)
CN L-Alanine, L-.alpha.-aspartyl-L-alanyl-L-.alpha.-glutamyl-L-phenylalanyl-L-arginyl-L-histidyl-L-.alpha.-aspartyl-L-serylglycyl-L-tyrosyl-L-.alpha.-glutamyl-L-valyl-L-histidyl-L-histidyl-L-glutaminyl-L-lysyl-L-leucyl-L-valyl-L-phenylalanyl-L-phenylalanyl-L-alanyl-L-.alpha.-glutamyl-L-.alpha.-aspartyl-L-valylglycyl-L-seryl-L-asparaginyl-L-lysylglycyl-L-alanyl-L-isoleucyl-L-isoleucylglycyl-L-leucyl-L-methionyl-L-valylglycylglycyl-L-valyl-L-valyl-L-isoleucyl-
CN Peptide .beta. (human amyloid)
CI MAN
SQL 42

SEQ 1 DAEFRHDSGY EVHHQKLVFF AEDVGSNKGA IIGLMVGGVV IA
====

TS AT: 16-19

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 138:12933

REFERENCE 2: 138:3442

REFERENCE 3: 138:3187

REFERENCE 4: 138:3139

REFERENCE 5: 137:383644

REFERENCE 6: 137:383298

REFERENCE 7: 137:368578

REFERENCE 8: 137:351513

REFERENCE 9: 137:350697

REFERENCE 10: 137:350680

FILE 'HOME' ENTERED AT 12:22:10 ON 06 JAN 2003